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MANAGEMENT OF OBESITY*

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Johannesburg

'I'm putting on weight, doctor; What ought I to do about it?' is a popular lament.

No generation has been so figure-conscious as the present. Never in recorded history has there been such hostile aversion to obesity. Till the end of last century the rotundity of John Bull was symbolic of health and virility. Today, tall, lanky Uncle Sam with his flat midriff is the figure of choice. His personality is graced for action and longevity.

When is one over-weight? When it is 10% above normal. What should the normal weight be? Roughly speaking it is (in lbs) 110, plus or minus the number of inches above or below 5 feet in height multiplied by 5½. Thus if height is 5 feet. 7 inches, weight should be approximately 148½ lb. If height is 4 feet. 10 inches, weight should be about 99 lb.

What makes us eat? In the normal individual *hypoglycaemia* plays a principal role. So long as the difference in the arterio-venous blood sugar is above 15 mg. per 100 c.c. there is no hunger. When it falls to zero, there is a craving for food. So, when the blood sugar level is low, appetite goes up. This 'glucostatic mechanism' is highly sensitive and believed to reside in the medio-ventral nuclei of the hypothalamus. These nuclei function as the 'appestat' or *governor* of food intake. Damage to these nuclei leads to enormous increase in appetite. The lateral nuclei of the hypothalamus stimulate appetite. When these are destroyed there is loss of appetite. It would appear that in the obese, the adrenal steroids provide the hypothalamus with perverted information demanding extra food in the midst of plenty.

Some believe, with the Freudians, that obesity is the result of arrested sexual development—the libido having stalled at the oral stage. Others maintain that it is the aftermath of parental neglect or overprotection in childhood, and others contend that it is a substitute for frustrations.

The question is often asked why some eat a lot and never gain weight. It would appear that the healthy body regulates its weight with remarkable nicety—the 'homeostatic mechanism', also situated in the brain stem. In the over-weight this mechanism has broken down. It has lost its subtlety.

The quest for a successful *treatment* of obesity has always overshadowed the search for its *causes*. In the over-weight, the 'will power' to eat rich foods is mightier than the 'won't' power.

Obesity is the half-way house between health and disease. According to statistics 1 out of every 4 persons is overweight.

Overweight is commonest during middle life, and is due to excess of calories. As we grow older metabolism slows down and fewer calories are needed. At middle age there is less desire for physical exercise. Gormandizing is an outlet for emotional tension, boredom, and frustration. We put on weight when we are inactive.

To fight 'dangerous curves' and to conquer the 'battle of bulges' one's mental climate must change. We all realize that a slimmer waistline means a longer life-line. The over-weight loves to be told that the cause of the trouble is 'glandular'. No one welcomes the verdict that he eats too much. The endocrine system is seldom at fault. The only glands involved are the salivary glands.

What harm does this extra weight do? Assuming one is 20 lb. over-weight, the body has to provide a fleet of blood vessels a mile long to feed it, and the heart is forced to pump continuously extra blood through a widely scattered territory of useless tissue. Life expectancy is thus reduced.

POPULAR OBESITY CURES

Thousands of starvationists do themselves irreparable harm. Fortunes are flung away on worthless nostrums

* A paper presented at the South African Medical Congress, Pretoria, October 1955.

consisting of tablets, pills, teas, liquids, and chewing gums—all alleged to reduce weight. On analysis, they are found to consist of laxatives, thyroid, pituitary, dinitrophenol, and amphetamine. Some of these pills and tablets are given a 'new look' by turning them out in technicolour. They are expensive, useless and even dangerous. The nostrum vendors also sell vibrators, rollers, girdles, vanishing creams, and bath salts. They gull the intelligentsia as easily as they fool the illiterate and credulous public by claiming cures without diet and without exercise. They claim to convert stouts into sylphs.

Rapid cures for obesity hit the headlines in newspapers and journals with remarkable regularity. We have today a glut of 'diets': 18-day, 11-day, 7-day, 2-day, and 'skip-a-day' slimming diets. We have the 'crisp-carrot-and-lettuce diet', the 'all-meat diet', the 'morning-noon and- night egg diet', the 'pineapple diet', the 'banana-and milk,' 'egg-and-spinach,' 'bread-and-butter', and 'potato-and-gravy' diets, and other freak diets fabricated by food faddists *ad infinitum*. The results are uniform. While the enthusiasm lasts, the pounds melt away; but after a few weeks the appetite is cloyed and the devotee reverts to old habits—first a nibble, then a gobble, and then it is 'good-bye diet'. In the end he confesses that all he has lost is the zest to continue with the magic diet.

All these diets are badly balanced and one-sided. In the long run amateur dieting is harmful and best left alone. There are no short cuts. No easy way is safe, and no safe way is easy. When we think of reducing we search for short cuts. Most of us never realize what poor losers we are until we try to lose weight. The motto of the overweight is 'eat, drink and be merry, for tomorrow we *diet*'; but tomorrow never comes. We know full well that we should look more *spick* if we had less *span*.

Some dieters feel weak and miserable when they lose weight. For them it is better to be fat and alive than svelte and half-dead!

SUCCESSFUL WEIGHT REDUCTION

To reduce successfully, one must be quite serious about it. One must be prepared to unlearn old habits and adopt new. It requires strength and determination. Weight reduction is the triumph of mind over platter. If the doctor gets full cooperation, the patient will lose weight as certainly as the sun rises in the east. The recruit must develop an abhorrence for succulent dishes. He must block his ears and shut his eyes to ridicule and commiseration. He must be prepared to stick to the new regime for the rest of his life. His resolve must never dissolve. His reward will be rich. He will be freed from his own fortress of fat, regain his physical vigour, and find it easy to bend down; unsightly bulges will disappear; he will be more alert, live longer, and enjoy many more years of happiness. He will have given nature a chance to undo the damage suffered by an outraged body.

In order to reduce, one should eat less, eat slowly and masticate thoroughly; have no more than 3 meals a day and no second helpings; eat nothing between meals and indulge in no nibbling and no alcohol.

Anorectics such as amphetamine may be necessary. When used with discretion they have proved a boon to the obese. They act as useful crutches and should be discarded as soon as possible. Pictures and charts should be used to help the patient to contrast his overweight with the normal weight for his height and age, and to realize the excess of weight he is carrying. For the obese, dinner in the morning, and breakfast at night is often good policy because he is less hungry in the morning, and does most of the eating after noon.

The over-weight should never eat when he is not hungry, or when he is emotionally upset or over-tired. Allow 5 hours between meals. He should rest when tired, sleep when sleepy, dress warmly, and avoid spirits, rich soups, sauces, spices, seasonings, and salad dressings.

Weight reduction is more than a cosmetic problem. Like the iceberg, the fat visible to the naked eye is but a fraction of the internal obesity that is invisible.

Fat children are usually a psychologic as well as a physiologic problem. They need discipline as well as diet.

In conclusion, the words of the Bard ring true:

> 'Make thy body less,
> Hence thy grace more:
> Leave gormandising and know
> That the grave doth gape for thee
> Thrice wider than for other men.'

BOOKS RECEIVED : BOEKE ONTVANG

A Manual of Practical Obstetrics. Third Edition. By O'Donel Browne, edited and largely re-written by J. C. Callagher.: Pp. viii+265, with 203 illustrations. 37s. 6d. net. Bristol: John Wright and Sons Ltd. 1956.

Experimental Physiology for Medical Students. Sixth Edition. By D. T. Harris, M.D., D.Sc., F.Inst.P., H. P. Gilding, M.A., M.D. and W. A. M. Smart, B.Sc., M.B., B.S. Pp. viii+289, with 252 illustrations and plate in colour. 30s. net. J. & A. Churchill Ltd. 1956.

Progress in Clinical Obstetrics and Gynaecology. By T. L. T. Lewis, M.B., F.R.C.S. (Eng.), M.R.C.O.G. Pp. viii+594, with 90 illustrations. 55s. net. London: J. &. A. Churchill Ltd. 1956.

A Modern Pilgrim's Progress for Diabetics. By Garfield G. Duncan, M.D. Pp. xiii+222. $2·50. Philadelphia and London: W. B. Saunders Company. 1956.

Diseases of the Chest. By H. Corwin Hinshaw, M.D., Ph.D. and L. Henry Garland, M.B., B.Ch. Pp. x+727, with 634 illustrations on 277 figures. $15·00. Philadelphia and London: W. B. Saunders Company. 1956.

Standard Atlas of Human Anatomy. One set (volumes I and II). By M. W. Woerdman, M.D., D.Sc. hon. causa (Oxon.). Volume I, 512 Figures, Volume II, 642 Figures. £4 0s. 0d. Amsterdam: Butterworth & Co. (Publishers) Ltd. 1955.

Pediatric X-ray Diagnosis. Third Edition. By John Caffey, A.B., M.D. Pp. xxiv+1059, with 2,264 illustrations on 1,267 figures. $28. Chicago: Year Book Publishers Inc. 1956.

Preparing for Motherhood. By Samuel R. Meaker, M.D. Pp. 196, with 19 illustrations. $2·00 post paid. Chicago: Year Book Publishers, Inc. 1956.

Gullan's Theory and Practice of Nursing. Seventh Edition. Revised by Marion E. Gould, D.N. Pp. xvi+244. Illustrations 2. 18s. net. London: H. K. Lewis & Co. Ltd. 1956.

The Control of Disease in the Tropics. By T. H. Davey and W. P. H. Lightbody. Pp. x+408, with 85 illustrations. £2 7s. 6d. net. London: H. K. Lewis & Co. Ltd. 1956.

South African Medical Journal

Suid-Afrikaanse Tydskrif vir Geneeskunde

EDITORIAL

THE INFLUENCE OF DIETARY FAT ON PLASMA LIPIDS

The view is today held to an increasing extent that the incidence of atheroma and coronary thrombosis is linked with the serum-cholesterol level, and that both are influenced by the quantity of fat in the diet. This view is based on epidemiological data concerning different national and social population-groups and the results of large-scale studies of diets and plasma lipids, and is supported by clinical and laboratory studies of individual subjects. A protagonist of the doctrine is Professor Ancel Keys, of the University of Minnesota, whose travels through the world in pursuance of his researches on the subject brought him last year to South Africa, where he worked for some weeks at Cape Town in Professor Brock's Clinical Nutrition Research Unit, in cooperation with the staff of the Unit, on the variations in these factors that are exhibited by the different ethnic groups in this country.

The researches in Cape Town were continued by Dr. Bronte-Stewart and his colleagues,[1,2] who showed that the widely differing inter-racial incidence of coronary heart-disease was associated with a parallel difference in mean serum-cholesterol levels, and that within each racial group these levels were highest in the highest economic plane and bore a parallel relationship to the intake of food rich in fat *of animal origin*. There was no correlation, they said, with the intake of vegetable fat.[2] There are, however, differences beside those of fat content in the diets of people in different economic planes in the various ethnic groups, and Bronte-Stewart *et al.* have found it necessary to make individual clinical and laboratory studies into the effects of various foodstuffs on the serum-cholesterol level under controlled conditions. A preliminary report on these researches has now been published.[3]

The studies reported were carried out on a few volunteers in whom the serum cholesterol (and its β-lipoprotein element) were measured as changes were made in their controlled diet—chiefly in its fat content. In Bantus accustomed to a low-fat diet and actually on a low-fat, low-protein and cholesterol-free diet,

VAN DIE REDAKSIE

DIE INVLOED VAN DIE DIEETVET OP DIE PLASMA-LIPIEDE

Dit word vandag meer en meer geglo dat daar 'n verband is tussen die voorkoms van slagaarvervetting en kroonslagaartrombose, en die serumcholesterolstand, en dat albei siektes deur die hoeveelheid vet in die dieet beïnvloed word. Hierdie mening is gegrond op die epidemiologiese gegewens aangaande verskillende nasionale en sosiale bevolkingsgroepe en die uitgebreide studies van diëte en plasma-lipiede, en word gesteun deur kliniese en laboratoriumstudies wat op indiwiduele persone gemaak is. Professor Ancel Keys van die Universiteit van Minnesota is 'n voorstander van hierdie mening. Sy reise om die wêreld in verband met sy navorsing op hierdie gebied het hom verlede jaar na Suid-Afrika gebring, en hy het 'n paar weke lank in Professor Brock se Clinical Nutrition Research Unit in Kaapstad saam met die personeel van die eenheid navorsingswerk gedoen op die veriasies wat die verskillende etniese groepe in hierdie land in hierdie faktore vertoon.

Dr. Bronte-Stewart en sy medewerkers[1,2] het die navorsing voortgesit, en het bewys dat die wyd-uiteenlopende rasseverskil in die voorkomssyfer van kroonslagaar-hartsiekte in verband staan met 'n ooreenstemmende verskil in die gemiddelde serum-cholesterolstande; dat, binne elke rassegroep, die cholesterolstande die hoogste was by die mees gegoede sosiale stande, en dat dit in verhouding ooreenstem met die eet van voedsel ryk aan vette van *dierlike oorsprong*. Hulle kon geen wederkerige verhouding insake die inname van plantvette vasstel nie.[2] Daar is egter ook ander verskille behalwe dié van die vetinhoud in die diëte van mense van verskillende ekonomiese stande in die verskillende etniese groepe, en Bronte-Stewart *et al.* het dit noodsaaklik gevind om indiwiduele kliniese en laboratoriumstudies te maak van die uitwerking van die verskillende voedselsoorte op die serum-cholesterolstand onder beheerde toestande. 'n Voorlopige verslag oor hierdie navorsing het nou verskyn.[3]

Die gerapporteerde studies was uitgevoer op 'n paar vrywilligers by wie die serum-cholesterol (en sy β-lipoproteïene-bestanddeel) gemeet was by elke verandering—hoofsaaklik insake die vet-inhoud—wat in hulle beheerde dieet gemaak is. By Bantoes wat aan 'n vet-arm dieet gewoond is, en wat by die proefneming 'n vet- en proteïen-arm, cholesterol-vry dieet moes hou,

the addition for a few days of 100 g. a day of beef dripping (tallow), or of butter, or of egg-fat (10 eggs a day) produced an increase in serum cholesterol (mainly an increase in β-lipoprotein). When the addition consisted of 100 g. a day of ground-nut oil or sunflower-seed oil, which contain a high proportion of unsaturated fatty acids (particularly linoleic acid), instead of an increase a decrease occurred in the serum cholesterol (and β-lipoprotein). A similar addition of *hydrogenated* ground-nut oil, however, resulted in an increase in these plasma lipids. Olive oil appeared to take an equivocal or intermediate position in its effect on the plasma lipids.

In 2 Europeans, who were actually suffering from coronary disease and were on a diet rich in animal fat, to which they were accustomed, and were giving a high serum-cholesterol figure, the addition of 100 g. of sunflower-seed oil or ground-nut oil produced a marked fall in serum-cholesterol (and in β-lipoprotein), which at once reverted to its original higher level when the addition was discontinued. In various experiments the addition of sunflower-seed oil caused this fall notwithstanding that the addition involved an increase in the total amount of fat that was being administered, and notwithstanding that there was no reduction of protein or cholesterol in the diet.

When sunflower-seed oil was fractionated into a saturated and an unsaturated portion, the addition to the diet of the saturated fraction produced an increase in the cholesterol figures, and of the unsaturated fraction a decrease (in a Bantu subject).

The addition to the diet of eggs, which, beside fat, contain protein and cholesterol, was very potent in raising the serum-cholesterol level in the Bantu subjects when the eggs were eaten boiled, but when they were scrambled or fried in sunflower-seed oil the increase in serum cholesterol was wholly or partly avoided.

Pilchard oil and seal oil, which contained a high proportion of unsaturated fatty acids, acted similarly to the vegetable oils in producing a fall in the serum-cholesterol level when added to the controlled diet.

These results reinforce the view that different natural fats and oils have different effects on the level of serum cholesterol, and that the variation is associated with the difference in the proportion of saturated and unsaturated fatty acids in their composition. The suggestion that certain unsaturated fats in the diet are not merely neutral in relation to plasma lipids, but have actually a positive action which leads to a reduction in the serum-cholesterol level, is of particular interest in view of the suggestion made by H. M. Sinclair[1] that the increase in recent years in coronary heart-disease is causally associated with a relative deficiency of certain unsaturated fatty acids such as linoleic and is dit bevind dat die serum-cholesterolstand (hoofsaaklik die β-lipoproteïen) verhoog is deur die byvoeg oor 'n paar dae van 100 g. bees-braaivet (harde vet) of van botter, of van eiervat (10 eiers per dag). Toe die bygevoegde voedsel egter vervang is deur 100 g. grondboontjie- of sonneblomsaadolie daagliks (wat 'n hoë gehalte van onversadigde vetsure, veral lynoliesuur, bevat), was daar in plaas van 'n vermeerdering inderdaad 'n vermindering in die serum-cholesterol (en β-lipoproteïene). 'n Soortegelyke byvoeging van *hidrogeneerde* grondboontjie-olie het egter 'n vermeerdering van hierdie plasma-lipiede tot gevolg gehad. Olyfolie het blykens die proefnemings 'n onsekere of intermediêre uitwerking op die plasma-lipiede.

By 2 Blankes wat inderdaad aan kroonslagaarsiekte gely het, en wat 'n dieet ryk aan diervette (waaraan hulle gewoon was) gehou het, en by wie die serum-cholesterolstand hoog was, het die byvoeging van 100 g. sonneblomsaad-olie of grondboontjie-olie 'n belangrike afname in die serum-cholesterol (en in β-lipoproteïne) bewerkstellig. Die verminderde serum-cholesterol het onmiddelik na sy vorige gehalte gestyg toe hierdie byvoeging gestaak is. By verskeie proefnemings het die byvoeging van sonnebloomsaad-olie hierdie daling veroorsaak nieteenstaande die feit dat die byvoeging 'n vermeerdering in die totale inname van vet beteken het, en dat die hoeveelheid proteïene of cholesterol in die voedselplan geensins verminder was nie.

Met die skei van sonneblomsaad-olie in versadigde en onversadigde dele, het die toevoeging van die versadigde deel tot die dieet van 'n Bantoe 'n toename in die cholesterol-syfers veroorsaak, en die toevoeging van die onversadigde deel het dit verminder.

Die byvoeg tot die dieet van eiers, wat behalwe vet ook proteïene en cholesterol bevat, het die serum-cholesterolstand by Bantoes belangrik vermeerder indien die eiers gekook was, maar toe dit as roereiers voorberei of in sonneblomsaad-olie gebak is, is die styging van die serum-cholesterol heeltemal of gedeeltelik vermy.

Sardynolie en robbe-olie, met 'n hoë verhouding van onversadigde vetsure, het dieselfde uitwerking gehad as die plantolies; toe hulle by die beheerde dieet gevoeg is, het die serum-cholesterolstand gedaal.

Hierdie resultate skraag die mening dat die verskillende natuurlike vette en olies verskillende uitwerkings op die serum-cholesterolstand het, en dat die variasie in verband staan met die verskil in die verhouding tussen die versadigde en die onversadigde vette in hul samestelling. Die voorstel dat sekere onversadigde vette in die dieet nie net neutraal is wat die plasma-lipiede betref nie, maar dat hulle inderdaad die serum-cholesterolstand positief kan verminder, is besonder belangrik met die oog op H. M. Sinclair[1] se onlangse bewering dat daar 'n oorsaak-verband is tussen die toename in die voorkomssyfer van kroonslagaarhartsiekte in die laaste jare en die betreklike tekort aan sekere onversadigde vetsure soos lynoliesuur en aragidoniese sure (wat respektiewelik 2 en 4 dubbel-bindings bevat) in die dieet van die gemeenskappe waar die siekte voorkom. Meer as 25 jaar gelede het Burr en Burr[5] aangetoon dat hierdie soort vetsuur, behalwe as

arachidonic acids (which respectively contain 2 and 4 double bonds) in the diet of affected communities. That this kind of fatty acid plays an essential role in animal metabolism in addition to merely supplying calories was shown by Burr and Burr[5] more than 25 years ago, and the suggestion that a dietary deficiency of it might be related to atherogenesis has been made before.[6]

Ancel Keys,[6] however, holds strongly that the evidence shows that atheroma is caused by an excess of total fats in the diet rather than by a deficiency of fats of a particular kind. He maintains for instance that in the USA, with its high incidence of coronary thrombosis, the *per capita* intake of linoleic acid is 'at least as high as that of many populations, as in Japan, who suffer relatively little from coronary heart-disease'.

The position, then, in regard to the role of dietary fats in relation to atheroma and coronary disease, seems today to be more uncertain than it was thought to be a few months ago. It then appeared that the factor responsible for a high incidence of the disease was a diet containing a high proportion of fats of any kind, and that the relatively simple remedy was to reduce this proportion. The results obtained by Bronte-Stewart and his collaborators now suggest that it is not merely the total quantity of fat in the diet that counts, but also the *kind* of fat that is eaten; that there are two classes of food fats, whose effects on plasma lipids are antagonistic to each other.[6] These authors emphasize the short-term nature of their experiments and the small number of subjects studied; and they refrain from wholly attributing coronary disease to any single cause.[3] Their results, however, are most suggestive and it is evident that the problems presented by this great and increasing cause of death cannot yet be regarded as finally solved. It seems that more information is needed about the physiological significance of the complex structures of the lipids found in biological materials and their importance in pathology.[7]

1. Bronte-Stewart, B., Keys, A. and Brock, J. F. (1955): Lancet, **2**, 1103.
2. Bronte-Stewart, B., Moodie, A. D., Antonis, A., Eales, L. and Brock, J. F. (1955): S. Afr. Med. J., **29**, 1151.
3. Bronte-Stewart, B., Antonis, A., Eales, L. and Brock, J. F. (1956): Lancet, **1**, 521.
4. Sinclair, H. M. (1956): *Ibid.*, **1**, 381.
5. Burr, C. O. and Burr, M. M. (1929): J. Biol. Chem., **82**, 587.
6. Keys, A. (1956): Lancet, **1**, 576.
7. Editorial (1956): *Ibid.*, **1**, 557.

bron van kalorieë, ook 'n essensiële rol in die dieremetabolisme speel, en nog vroeër is dit voorgestel dat 'n tekort daarvan in die dieet moontlik met die ontwikkeling van slagaarvervetting gekoppel kan word.[6]

Ancel Keys[6] is egter oortuig daarvan dat slagaarvervetting volgens deeglik bewese feite veroorsaak word deur 'n oormaat van die totale vetstowwe in die dieet, en nie soseer deur 'n tekort aan bepaalde soorte vette nie. Hy verklaar byvoorbeeld dat die per capita inname van lynoliesuur in die VSA met sy hoë voorkomssyfer van kroonslagaartrombose ‚ten minste net so hoog is as dié van baie ander nasies, byvoorbeeld die Japanners, wat betreklik min aan kroonslagaarhartsiekte ly'.

Dit blyk dus dat die posisie insake die invloed van die dieetvette op vervetting en kroonslagaarsiekte vandag meer onduidelik is as wat ons 'n paar maande gelede gemeen het nie. Toe het ons gedink dat die faktor wat verantwoordelik is vir die hoë voorkomssyfer van hierdie siekte, 'n dieet met 'n groot hoeveelheid vette van alle soorte is, en dat dit betreklik maklik verhelp kan word deur die vermindering van die vetinname. Die uitslae van Bronte-Stewart en sy medewerkers se navorsing dui nou daarop dat dit nie bloot die totale hoeveelheid vet in die dieet is wat belangrik is nie, maar dat die *soort* vet wat geëet word ook van belang is; en dat daar twee soorte voedselvette is wie se uitwerking op die plasma-lipiede teenstrydig met mekaar is.[6] Hierdie skrywers benadruk die feit dat hulle navorsing oor 'n kort tydjie geskied het en dat hulle die proefnemings op 'n betreklik klein groep persone uitgevoer het. Hulle wil nie die ontwikkeling van kroonslagaarsiekte geheel en al aan 'n enkele oorsaak toeskryf nie.[3] Hulle resultate is egter veelseggend en dit is duidelik dat die probleme wat hierdie groot en steeds toenemende doodsoorsaak oplewer nog nie as finaal opgelos beskou kan word nie. Verdere inligting is nodig aangaande die fisiologiese belang van die ingewikkelde samestellings van die lipiede in biologiese stowwe, en aangaande hul belang in die patologie.[7]

1. Bronte-Stewart, B., Keys, A. en Brock, J. F. (1955): Lancet, **2**, 1103.
2. Bronte-Stewart, B., Moodie, A. D., Antonis, A., Eales, L. en Brock, J. F. (1955): S. Afr. T. Geneesk., **29**, 1151.
3. Bronte-Stewart, B., Antonis, A., Eales, L. en Brock, J. F. (1956): Lancet, **1**, 521.
4. Sinclair, H. M. (1956): *Ibid.*, **1**, 381.
5. Burr, C. O. en Burr, M. M. (1929): J. Biol. Chem., **82**, 587.
6. Keys, A. (1956): Lancet, **1**, 576.
7. Redaksie (1956): *Ibid.*, **1**, 557.

SOUTHERN AFRICAN CARDIAC SOCIETY

At a meeting of the Cape Province section of the Southern African Cardiac Society, held at Groote Schuur Hospital, Cape Town, on the evening of 12 April 1956, with Dr. Maurice Nellen in the chair, the following cases were shown:

1. *Dr. Maurice Nellen.* A case of heart disease of unknown etiology. ? Aberrant right coronary artery from pulmonary artery. ? Familial cardiomegaly. ? Fibro-elastosis. Illustrated with sound traces and phelbogram.
2. *Dr. V. Schrire.* A case of myocarditis associated with pulsus alternans and a discussion of the mechanism of pulsus alternans. Illustrated with sound and pulse tracings.
3. *Dr. L. Vogelpoel.* A case of Fallot's 'Trilogy' emphasizing the value of auscultation both in the diagnosis and in the assessment of the adequacy of pulmonary valvotomy. Fully documented with sound tracings taken before and after valvotomy.
4. *Dr. V. Schrire.* A case of Fallot's 'Trilogy' after successful pulmonary valvotomy the day before. Fully illustrated with sound tracings and pressure recordings during operation.

HYPOVENTILATION: ITS DANGERS IN GENERAL ANAESTHESIA

H. H. SAMSON, *M.B.E.*, M.R.C.S. (LOND.), L.R.C.P. (ENG.)*

Johannesburg

Hypoventilation is the clinical state which results from a diminution in the rate of exchange of the respiratory gases between the blood and the alveolar air. The oxygen content of the blood and consequently of the tissues is reduced. This is conveniently referred to as *hypoxia*. The CO_2 content of the blood is increased, and this is known as *hypercapnia*.

While these two hazards are almost always associated in hypoventilation occurring during anaesthesia, the effects of each can be studied in the absence of the other under experimental conditions. Hypoxia has been studied clinically as the cause of mountain or altitude sickness. As a result of oxygen lack, the respiratory centre is reflexly stimulated *via* the chemoceptors. Unconsciousness ensues if the lack of oxygen supply to the brain is gross enough. The effects of hypercapnia can be observed in the absence of hypoxia by asking a subject to breathe in and out of a re-breathing bag to which an adequate amount of oxygen is delivered, but without soda lime in the circuit to absorb the CO_2. The respiratory centre is stimulated in this case by an overdose of its normal stimulant. Unconsciousness occurs from the anaesthetic property of CO_2 when its proportion in the inspired gases is 10% (Clements[1]).

During quiet breathing in adult subjects the amount of air inspired or expired measures 350-500 c.c.; this is the *tidal volume*. *It ensures an adequate oxygen intake and CO_2 output*, because this volume of air expands the lung alveoli and thereby provides an area of pulmonary epithelium sufficient to enable the necessary diffusion of the respiratory gases. If this area of epithelium is reduced beyond a certain minimum, by a reduced tidal volume, it becomes inadequate for efficient respiration; in other words, the oxygen intake will be reduced below what the body requires, and the CO_2 output will be restricted. Thus hypoventilation leads inevitably to hypoxia and hypercapnia.

General anaesthesia is an abnormal condition, a temporary disease produced by the anaesthetist in the process of abolishing the perception of painful stimuli by means of narcotics (known as anaesthetic agents). *Its pathological severity can be estimated by the degree of hypoventilation which is allowed to occur.*

'The anaesthetist ordinarily over-estimates the extent of ventilation. In a number of observations with the carbon dioxide analyser, *tremendous retention* of CO_2 was demonstrated in patients to whom the anaesthetist felt he was providing adequate ventilation' (Cullen[2]).

CAUSES OF HYPOVENTILATION

The causes of hypoventilation may be classified as (1) local, (2) central and (3) general.

1. *Local causes* include diseases of the lungs, e.g. emphysema, atelectasis, fibrosis. Other local causes are obstruction of the airway, excessively narrow intratracheal tubes, abnormal positioning, e.g. the extreme Trendelenburg or the jack-knife position, and faulty apparatus such as inefficient expiratory and to-and-fro valves or badly fitting face-masks. An assistant leaning on the patient's trunk is another possibility, as is excessive retraction of the costal margin.

2. *Central causes* are those producing paralysis of the respiratory centre. They include the anaesthetic agents, with or without premedicating drugs.

3. *General causes* are drugs producing muscular relaxation, and hence paralysis of the muscles of respiration.

HISTORY OF ANAESTHETIC METHODS

In the days of the rag-and-bottle method of anaesthesia, most of the CO_2, including the concentration necessary to stimulate the respiratory centre, was lost. It went unhindered into the surrounding atmosphere. Without this stimulus, it was difficult to ensure a depth of respiration which would convey enough of the anaesthetic agent to the blood stream, especially when the agent was only mildly potent. Hence the open method gave way to the semi-open—the face mask was covered with towels, so that the concentration of the agent in the inspired air would be increased, when of course CO_2 began to build up simultaneously.

The introduction of the re-breathing bag followed logically. By the semi-closed technique the stage of induction could be shortened and anaesthesia could be more easily maintained. Oxygen administration was also facilitated. Finally, the closed circuit method was evolved in an endeavour to control the amount and proportion of the gases respired. The soda-lime canister became an important part of the apparatus, as the danger of CO_2 accumulation was at once apparent.

The Muscle Relaxants. Before the advent of the muscle relaxants, muscular resistance added considerable difficulty to abdominal surgery. Forcible retraction of the abdominal muscles and peritoneum was often responsible for post-operative collapse. If deep anaesthesia was maintained for a lengthy period, the ill-effects of hypoventilation were superadded, caused by depression of the respiratory centre. Nowadays, muscular resistance is abolished as if by magic by the injection of a muscle relaxant, but post-operative collapse may still occur, especially after or even during long operations. The reason is that the relaxants paralyse the respiratory muscles peripherally. *They are probably the most potent, and also the most dangerous, cause of hypoventilation.*

The use of muscle relaxants has been accompanied by an increase in morbidity and mortality, just as occurred with the introduction of the intravenous anaesthetics. The increased dangers are due to lack of appreciation

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of the hazards of hypoventilation produced by these potent drugs.

The wounded animal shot with the curarized arrow of the South American Indian dies immediately from the effect of a massive dose of curare[3] on the heart muscle. However, when the dose is smaller, the unfortunate animal often dies a lingering death from paralysis of the respiratory muscles, i.e., from hypoventilation. Consider the exquisite torture which must be experienced by a conscious animal when the healthy respiratory centre is not able to invoke a response to the most powerful analeptic in existence—carbon dioxide. A similar situation is created by the inept anaesthetist who curarizes his patient and then permits him to hypoventilate, except that the narcotized patient is spared the sufferings of the conscious animal.

The full significance of the distress produced by hypoventilation cannot be appreciated by a healthy person. One may get some idea however by voluntarily hypoventilating or by being strapped into a very tight corset for 1-2 minutes.

CO_2 ACCUMULATION—HYPERCAPNIA

When a patient is deeply anaesthetized and allowed to hypoventilate for a long period, one has observed that he will remain unconscious for a considerable time after the effect of the anaesthetic agent has worn off. This is due to the narcotic property of the accumulated CO_2. This effect was known long before the discovery of modern anaesthetics. Italian surgeons used to put their patients into small grottos before operation. The small opening in the vault was insufficient for satisfactory ventilation, and after many hours the patient became stupefied by his own CO_2. Breathing in and out of a rubber bag (a re-breathing bag) will reproduce a similar situation in a few minutes.

In small amounts CO_2 stimulates the respiratory centre. In strong concentrations, however, it is a very powerful cardiac poison. Prolonged exposure to even moderate amounts can cause myocardial damage, often of a serious nature. The pulse is usually full and bounding in commencing hypercapnia. Twitchings and convulsions may occur, but these are often masked by the anaesthetic and the relaxants.

OXYGEN LACK—HYPOXIA

Following anaesthesia in which hypoxia is permitted to be the predominating extraneous factor, the patient on recovering may display some of the manifestations of mountain sickness—restlessness, garrulity, shivering and various degrees of cyanosis. He may complain of severe headache or abdominal pain. The pulse is often rapid and of poor volume. On the other hand, should the hypoxia have been complicated by hypercapnia—which is usually the case, but at a later stage—these signs and symptoms will be masked if the resulting circulatory collapse is gross enough.

The open and semi-open methods do not include the use of the re-breathing bag, and are therefore not complicated by the use of the muscle relaxants, but collapse can occur if the anaesthesia is excessively profound or prolonged. Hypoventilation results from depression of the respiratory centre. If a relaxant *is* used, a bag is essential, for it provides the only effective means of administering oxygen under pressure for combating acute hypoxia. The semi-closed and closed circuit methods permit the use of the muscle relaxants and thereby become correspondingly more dangerous in the presence of uncontrolled anaesthesia (no attention to the tidal volume).

Circulatory collapse ascribed to hypoventilation is therefore obviously due to the degree and duration of the hypoxia and hypercapnia produced, and is aggravated by (1) the type of anaesthetic agent (the effects will be more rapidly manifested with those that have a powerful depressing effect on the respiratory centre, such as thiopentone, cyclopropane and chloroform) and (2) the type of circuit used. The closed method is potentially more dangerous, because the re-breathing bag tends to allow accumulation of CO_2, *and its progressive displacement of oxygen.* Furthermore, gases are used in small amounts in this method—c.c. as compared with the litres used in the semi-closed method, and any leakage has thus a proportionately greater effect. Such leakage, occurring during expiration, implies that the bag does not receive the full tidal volume. Inspiration is consequently defrauded by the amount lost and hypoventilation commences. The soda-lime canister will take longer to warm up and the unwary anaesthetist is apt to interpret this as an indication of its high potency. The truth of the matter is that the soda-lime is actually doing less work, since it is not dealing with the full tidal volume. Its continued coolness should warn the alert administrator to check up on his apparatus immediately.

POST-ANAESTHETIC COLLAPSE FOLLOWING HYPOVENTILATION

A typical case history of this grave complication of hypoventilation is as follows: The patient is in good health, except for the complaint which requires a surgical procedure of long duration. Hypoventilation is allowed to occur, and hypoxia and hypercapnia develop insidiously. The colour remains deceptively pink—deceptive because it is the result not of adequate oxygen content of the blood, but of the excessive dissociation of oxygen from the blood to the tissues on account of the high CO_2 content. 'Too many anaesthetists for too long have been satisfied with having the patient "pink." One should not be lulled into complacency about the patient's ventilatory sufficiency on the basis of the oxygen supply alone' (Cullen[5]).

The blood itself becomes dusky, as may be apparent to the watchful surgeon if there is free bleeding. The blood pressure rises, and the pulse may become full and bounding. If the surgeon enquires after the patient's condition, he may be informed by the unsuspecting anaesthetist that everything is going well. The latter has been 'lulled into complacency' by the pink colour and the strong pulse. In marked hypoventilation, cardiac arrhythmia may occur.

Towards the end of the operation, collapse may set in, but is often delayed until the patient has been taken back to the ward. Here the sister observes that he lies limply

in bed, as if concussed. His colour is sickly pale and the breathing very shallow. The eyelids are slightly open, displaying dilated pupils. The pulse is rapid and thready and the blood pressure has fallen alarmingly. All reflexes may be absent.

The operating team is hastily summoned, and is often nonplussed by the suddenness of the collapse. '*But he was so well when he left the theatre, Sister! Was the jaw supported on the way down?*'

Very little blood may have been lost during the operation, and very little trauma may have been inflicted, nevertheless a pint of blood, or even 2 pints, is immediately ordered, and cardiac stimulants injected. *Neither the blood nor the stimulants exert any beneficial effect and the blood especially may well be harmful to the failing circulation in a severe case.* It is as illogical to transfuse a case of post-anaesthetic collapse as it would be to transfuse one of hyperglycaemic or hypoglycaemic coma.

CO_2 is sometimes administered, but it is absolutely contra-indicated, for the patient is already suffering from CO_2 intoxication and, furthermore, the paralysed respiratory centre is no longer able to respond.

Prevention

This implies prevention of the hypoventilation:

1. *Effective Apparatus.* Anaesthetists must in the first instance ensure that the more obvious causes, such as obstruction of the airway or leakages, are absent. An intratracheal tube minimizes the margin of error presented by the dead space, and also the additional hazard of obstruction.

2. *Maintenance of the Tidal Volume.* One must be able to estimate the tidal volume by the excursions of the thoracic cage and the abdomen, and of the re-breathing bag. This is not easy, but the required ability can soon be attained by practising assessment of one's own tidal volume with a re-breathing bag and a spirometer. If a decrease in the patient's tidal volume is noticed, it should be corrected immediately. Unless this is done, a lesser volume of gases is inspired (including less oxygen), and a lesser volume is expired (including less CO_2). Hence CO_2 must accumulate in the blood. Some administrators do not perceive the inexorable logic of this train of events. They do not comprehend how CO_2 accumulates in the blood when all the expired gases are released freely to the surrounding atmosphere. Nor do they appreciate how hypoxia can develop when the inspired gas is pure oxygen—forgetting that its volume is now insufficient to fill all the alveoli. However desperately the body requires more oxygen, it cannot absorb its requirements from the super-saturation of the volume of blood flowing to a limited area of lung tissue.

As soon as a diminution in the tidal volume is observed, mechanical or manual compression of the bag must be instituted. I consider the manual method easier and safer, provided one has learned to estimate the degree of compression required to raise the tidal volume to its correct level.

3. CO_2 *Absorption.* Soda-lime plays a most important part in closed-circuit anaesthesia. It is the only means of eliminating the excess CO_2. However, it is unwise to rely implicitly on the claims of the manufacturers concerning the life of the soda-lime. During lengthy procedures it is wise to use as many as 3 canisters, changing from one to the next as soon as it gets warm, not excessively hot—say, every 15-20 minutes. By so doing the accumulation of CO_2 from exhaustion of the soda-lime is prevented. The earliest detectable sign of hypercapnia is often a slight rise in the blood pressure, and this should be measured with regular frequency. Occasionally, the CO_2 content of the re-breathing bag should be measured with a CO_2 analyser as a confirmatory check.

4. *The re-breathing bag* is a potential reservoir of CO_2. It should not be very large. The bigger the bag, the greater the risk of CO_2 accumulation in it, and the greater the delay and difficulty in filling it with oxygen, when pressure is urgently required. A capacity of about 2 litres is quite adequate. Those who are not familiar with the size of tidal volumes can easily learn it by grasping a portion of the re-breathing bag so as to produce an hour-glass effect. The gas should now be made to flow into the upper compartment at the rate of the required volume in exactly 1 minute. Thus after 1 minute at the rate of say 500 c.c. per minute the compartment will either be over-distended or incompletely filled, and after a little practice the correct position of the hour-glass constriction will be found.

Once again it must be stressed that it is not sufficient to treat a hypoventilating patient by disconnecting him from the re-breathing bag and allowing the expired gases to pass unhampered into the ambient atmosphere. With a diminished tidal volume, less CO_2 will be excreted and more will be retained in the blood. Oxygen intake will also be lessened. The only logical method available is to retain the bag and compress it to maintain the tidal volume while absorbing the CO_2 by soda-lime.

5. *The muscle relaxants.* Long-acting relaxants are responsible for longer periods of hypoventilation and are therefore potentially more dangerous than the short-acting group. For this reason, mainly, I prefer Scoline. Some of my colleagues refuse to use it because there is no antidote. They say they feel safer with the longer-acting relaxants because an antidote is available—prostigmine. I consider this to be faulty reasoning. Sensitivity to Scoline is extremely rare—I have encountered 2 cases in over 3,000. Apnoea lasted 15 and 20 minutes respectively. In such cases, a sure antidote is in fact available—oxygen under pressure, and this was given with good effect. This type of apnoea must not be confused with that resulting from excessive loss of CO_2 or with a peculiar delay in the respiration probably due to irritation by the intratracheal tube. Secondly, I feel that the anaesthetized patient should be maintained as far as possible in his normal physiological state. The use of prostigmine (which must be accompanied with atropine) seems to be an example of treating an abnormal condition with an abnormal drug. Surely the administration of an antidote for an excessively-acting drug should not become an established routine. One may have to seek an antidote to the antidote! In any case, Scoline offers no such difficulty. The last fractionated dose is given to facilitate closure of the peritoneum, and has worn off long before the skin wound is closed.

Treatment

1. *Moderate collapse.* Provided that the patient was reasonably healthy before the anaesthetic ordeal, all that is necessary is to administer oxygen at the rate of 2-3 litres per minute through a clear airway, but with no bag. The foot of the bed should be raised and the patient kept warm. No narcotics should be given for the relief of questionable pain. Methedrine, 0·75–1 c.c. intramuscularly administered, at quarter hour intervals, may be of benefit.

2. *Severe collapse.* Severe cases must be treated on heroic lines. When the blood pressure has dropped alarmingly, the immediate danger is the circulatory failure and the consequent hypoxia of the vital parenchymatous organs. The aim of treatment is two-fold, viz. (*a*) restoration of circulation, and (*b*) effective administration of oxygen and elimination of the accumulated CO_2.

(*a*) *Restoration of circulation.* Add 4 c.c. of Levophed to a 1,000-c.c. vacolitre of fluid (5% glucose in water). To avoid the danger of tissue-sloughing, which may occur at the site of injection, insert a needle (or a cannula through a cutdown) into a vein, and connect up to the vacolitre. Raising of the limb to above the level of the heart may also help in the prevention of tissue-gangrene. The backward seepage of nor-adrenaline will also be minimized. Start the drip at about 40 drops per minute and regulate it according to frequent blood-pressure measurements. Usually, after 2-3 minutes, a rate of about 20 drops per minute will suffice to maintain the blood pressure at a safe level (which will obviously vary with the pre-operative level).

(*b*) *Effective administration of oxygen and elimination of the accumulated CO_2.* At the same time, administer oxygen at 0·75–1 litre per minute by means of a face-piece, with the expiratory valve almost completely closed. The CO_2 is absorbed by fresh soda-lime in a canister interposed between the patient and the re-breathing bag, which is vigorously and regularly compressed. The tidal volume should be raised to, say, 650 to 700 c.c. so as to hasten the excretion of CO_2. The apparatus at the same time should be checked for any leakage. It will be observed that the canister becomes abnormally hot in a matter of minutes and must therefore be changed frequently.

The pumping-in of 8-10 litres of oxygen per minute is not an effective method. The expiratory valve has to be kept far too open and, as a result, the full tidal volume cannot be attained with any degree of accuracy.

The Oxford Vaporiser is an apparatus which can be used effectively if no soda-lime is available. There is a 2-way valve which allows oxygen to be forced into the lungs but prevents any of the expired gases from re-entering the bag. This is a very useful apparatus and can be effectively used for maintaining the tidal volume.

DISCUSSION

It should be remembered that the anaesthetic agents were discovered and used by those who must have been well aware of their potency in the presence of normal ventilation. The abolition of pain was always the prime purpose. When these agents are abused, especially for the purpose of acquiring relaxation, extraneous factors such as hypoxia and hypercapnia frequently intrude. *The aim of general anaesthesia* should be to render a patient insensitive to painful stimuli with a minimal disturbance of his physiology. Muscle relaxants should be added when it is necessary to abolish muscular rigidity, including laryngeal spasm, and local anaesthetics when pain perception cannot be prevented by moderate amounts of anaesthetic agents.

The danger of anaesthesia should be measured in terms of the degree of hypoventilation, i.e. of hypoxia and hypercapnia. The anaesthetist must watch constantly and carefully for the signs of hypoventilation. These include a rising blood pressure and pulse rate, occasionally cardiac arrhythmia, and muscular rigidity.[6] They do *not* include alterations in the pupillary and other reflexes, which are an indication of the extent of overdosage with the anaesthetic agent.

In this connection, one must not confuse the effects of an anaesthetic agent with the effects of any associated hypoventilation, as is still commonly done in some of our text-books. Recent advances in anaesthesia have been revolutionary, and it might be advisable to re-assess findings published as long ago as 1911. In that year, for instance, Goodman Levy[7] stated that when cats under light chloroform anaesthesia were stimulated, they developed ventricular fibrillation. Surely one of the first rules of experimental human physiology is not to accept the evidence of animal experiments as being applicable to man! Levy omitted to describe exactly how his cats were anaesthetized. It would appear that they were not premedicated, and they may have been somewhat excited and uncooperative! They may even have hyperventilated! With the removal of the normal stimulus to breathing, they may then have taken in less oxygen. It is also possible that the ventricular fibrillation may have been due to factors only remotely related to the specific anaesthetic agent employed.

Even modern writers seem prone to make unwarrantable assumptions. Clements[8] believes that cardiac irregularities may occur with cyclopropane. In this he is supported by several other modern writers,[9] but it is the author's firm conviction *that the agent is being made the scapegoat for undetected hypoventilation.*

CONCLUSIONS

An appreciation of the nature and amount of the gases in the re-breathing bag is essential for the efficient administration of a general anaesthetic. It must be remembered that in an individual at rest any diminution of the tidal volume will reflect on the amount of oxygen absorbed and CO_2 expired.

Hypoventilation due to tampering with the tidal volume, whether this is effected with narcotics that depress the respiratory centre or the relaxants that so grossly interfere with the normal excursion of the thoracic wall, is a sure method of producing hypoxia and hypercapnia.

Many of the weird, worrying and so-called inexplicable

phenomena associated with general anaesthesia, including arrhythmia of the heart, and post-anaesthetic collapse, can be attributed to the extraneous factors—hypoxia and hypercapnia. In collapse from this cause blood transfusions are contra-indicated.

The anaesthetist should be constantly on the alert for the prevention of hypoventilation—not only during anaesthesia, but also when the patient has been returned to the ward.

No matter what method is adopted, hypoventilation may occur immediately the patient becomes anaesthetized. *The administrator should therefore strive at all times to maintain the tidal volume, which is the all-important factor in general anaesthesia.*

I wish to express my thanks to Mr. M. Arnold, F.R.C.S.E. for his invaluable assistance in the preparations of this article.

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THE REACTION OF PATIENTS WITH TYPHOID FEVER TO THE ADMINISTRATION OF ASPIRIN

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For many years the medical staff of the City Hospital for Infectious Diseases, Cape Town, have recognized the occurrence of a severe reaction following the administration of aspirin to patients with typhoid fever. This reaction, characterized by a sudden fall in temperature to subnormal levels, collapse, sweating and bradycardia, is far in excess of the usual antipyretic and diaphoretic action of the drug. When a case was encountered recently in Groote Schuur Hospital, reference to the available literature on typhoid fever failed to reveal any detailed record of a reaction of this kind.

Vincent and Muratet (1917) made the observation that as little as 1 g. of Cryogenine (phenylsemicarbazide) produced collapse with sweating and bradycardia in some patients with typhoid fever, and Nelson and Pijper (1951) decry the use of aspirin and other antipyretics because of the transient fall in temperature they produce by sweating, which leaves the patient considerably weakened. Knight *et al.* (1950) found bizarre temperature charts in patients with typhoid fever who were given antipyrine or quinine. Other authors (Gay, 1918; Byam and Archibald, 1922; Osler and Macrae, 1920; Ker, 1911; Harries and Mitman, 1953) recommend that antipyretics be withheld in typhoid fever, without giving reasons for this advice.

Aspirin or an analogue is frequently given in typhoid for the fever, headache or other pain.

This study was undertaken in an attempt to ascertain the frequency and severity of this reaction.

Material

The case records of all patients with typhoid fever admitted to Groote Schuur Hospital from January 1952 to May 1955, inclusive, were studied. There were 43 cases. Of these, 8 had had aspirin or aspirin-containing drugs administered at some stage during their stay in hospital. In addition one case (Case 4) is included, admitted in 1949, which showed the reaction and to which my attention was drawn.

RESULTS

(*a*) *Patients to whom aspirin was administered and who showed the reaction*

There were 5 such cases, 2 of which (cases 1 and 2) I saw during the acutely-ill stage.

Case 1. B.D., a Coloured male aged 20 years, was admitted to the hospital on 6 May 1955, complaining of headache, malaise and feverishness which had been present for 4 days. On the day before admission he had been given 2 'APCs' by his private doctor and profuse sweating, weakness and collapse occurred shortly thereafter. On admission his temperature was 103°F, he looked ill and toxic, and his abdomen was distended. Chloramphenicol therapy was started after blood cultures had been taken. His condition remained unchanged for 3 days, and at 6.35 p.m. on 10 May 2 tablets of aspirin, phenacetin and codeine were given for his severe headache. This was followed by a rapid fall in temperature from 103°F, reaching a nadir of 95·6°F at 8.30 p.m. When seen at this stage the patient was very pale, collapsed and prostrated, with profuse sweating and bradycardia, and was gravely ill. The blood pressure had fallen from its previous level of 130/80 mm. Hg to 110/60 mm. Hg. A tablespoonful of sodium bicarbonate was given in an attempt to accelerate the elimination of the aspirin, and the pulse rate and temperature were recorded at half-hourly intervals until these had returned to their former level and the patient regained his former clinical state. This took approximately 7 hours. Subsequently the patient responded well to therapy and was discharged on 17 May. The temperature chart is reproduced in Fig. 1.

Case 2. J.R., a Coloured male, 22 years of age, was admitted to the hospital on 2 May 1955. He complained of headache, malaise and abdominal discomfort of gradual onset starting 3½ weeks before admission. On admission he was extremely ill and his temperature was 98·8°F. This had risen to 102°F by the following morning. At 9.30 p.m. on 3 May, two 5-gr. tablets of aspirin were administered for abdominal pain and headache. The patient lapsed rapidly into a state of profound asthenia and prostration, with sweating and bradycardia, and the temperature fell to 95·6°F within 2 hours. These symptoms subsided and the temperature returned to its former level after a total of 5½ hours.

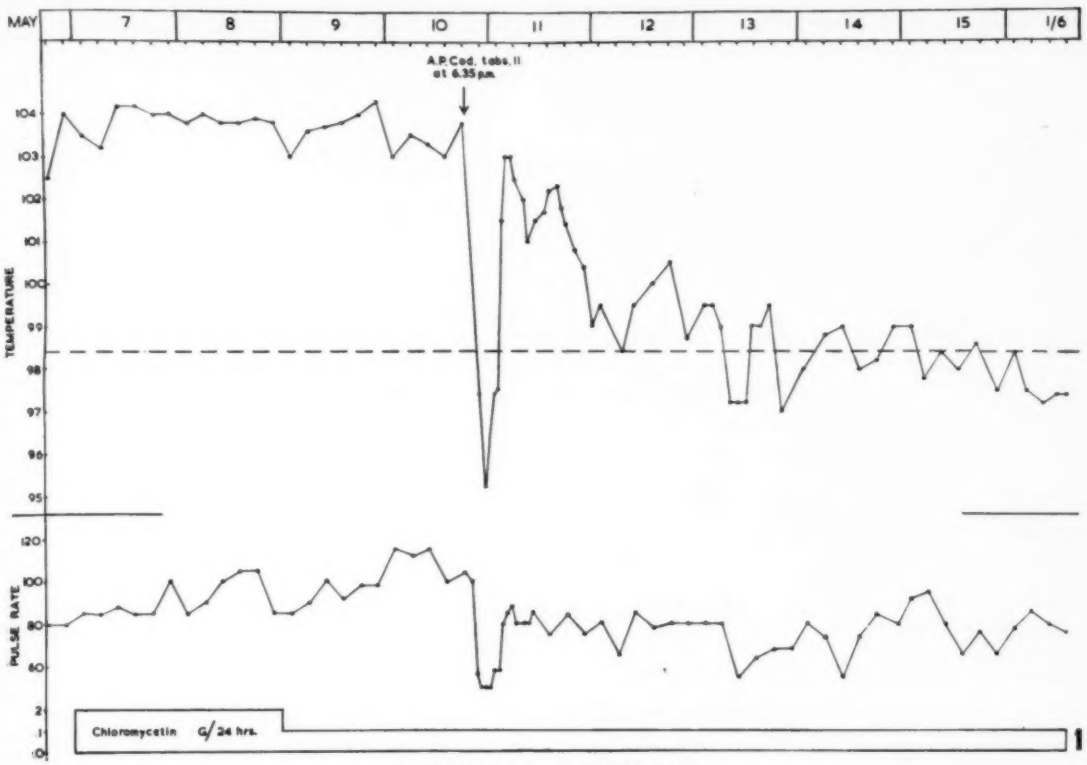

Fig. 1. Temperature chart of case 1.

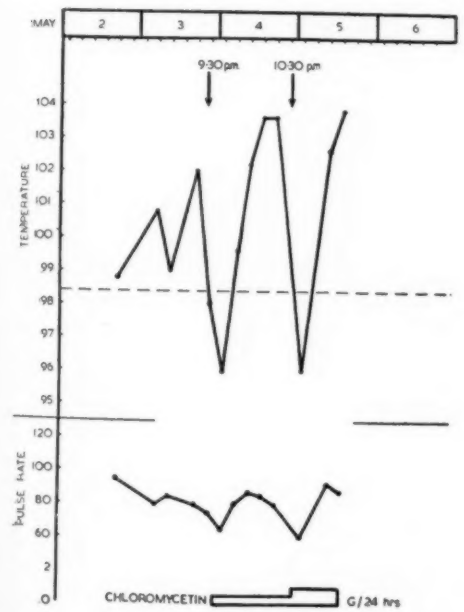

Fig. 2. Temperature chart of case 2.

On 4 May at 10.30 p.m. 2 tablets of aspirin were given with identical effect. He made an uneventful recovery on chloramphenicol. The temperature chart is reproduced in Fig. 2.

Case 3. K.L., a Coloured female of 21 years, was admitted on 16 April 1955 for investigation of symptoms of headache, feverishness and abdominal pain which had been present for 1 week before admission. When admitted she looked ill and apathetic and had a temperature of 102°F. A soft spleen was palpable. On 16 April 2 tablets of aspirin, phenacetin and codeine were given for the headache, with no apparent ill-effect and some relief of the headache. On 18 April, 15 gr. of aspirin were given at 6.35 p.m. This was followed by a sharp drop in temperature to 95°F, with sweating, collapse and bradycardia. After a total of 5½ hours she had regained her former state and made a subsequent complete recovery on chloramphenicol. The temperature chart is reproduced in Fig. 3.

Case 4. A.H., a White male of 29 years, was admitted on 2 July 1951, complaining of symptoms of headache, feverishness and general malaise for 5 days which had failed to respond to penicillin therapy. He appeared ill and toxic, with a high temperature and moderate splenomegaly. On 8 July 2 tablets of aspirin, phenacetin and codeine were administered and a sharp fall in temperature to 96°F followed. It was associated with a gravely collapsed state which caused considerable concern. After a total of 6 hours he had made a good clinical recovery from the acute episode, but his temperature took 12 hours to return to its previous level. The patient made a good recovery without specific antibiotic therapy. The temperature chart of this patient is reproduced in Fig. 4.

Case 5. J.S., a White female of 15 years, was admitted on 30 January 1952, on the 8th day of her illness. She was accompanied by a domestic temperature chart that had been kept by her mother, which showed an abrupt fall in temperature from

2

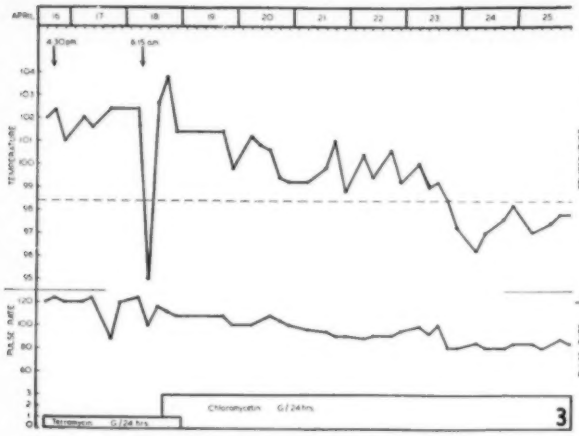

Fig. 3. Temperature chart of case 3.

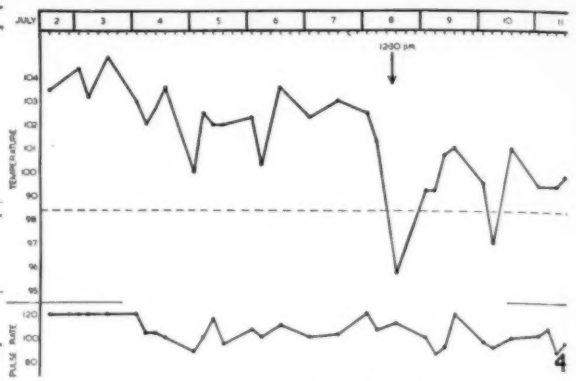

Fig. 4. Temperature chart of case 4.

106°F to 96°F following immediately upon the administration of 2 'gelonidas' on the day before admission. This, in the words of her private doctor in an accompanying letter, was associated with a 'grave deterioration in her clinical state'. She was treated with chloramphenicol, with defervescence on the 4th hospital day and subsequent complete recovery.

(*b*) *Patients to whom aspirin was administered without apparent ill effect*

There were 5 such cases, details of which are summarized in Table I. It will be seen that in 2 of these cases the drug was administered after defervescence had taken place.

(*c*) *Patients who showed sudden collapse with low temperature, where there was no good evidence of aspirin having been administered.*

There were 3 such cases. In 2 of them the collapse and hypothermia followed massive intestinal haemorrhage and responded well to whole-blood transfusion. In the 3rd case transient collapse with subnormal temperature was observed on the 2nd hospital day without any obvious cause being found. The ward sister, when questioned 4 weeks later, could remember aspirin having been given to the patient, but was uncertain of the precise time of administration of the drug.

(*d*) *Patients who did not receive aspirin and in whom there was no unexpected fall in temperature or deterioration in clinical state*

There were 31 cases in this remaining group.

TABLE I. CASES IN WHICH ASPIRIN OR ASPIRIN-CONTAINING DRUGS WAS GIVEN WITHOUT APPARENT ILL-EFFECTS

Case	*Age*	*Race*	*Sex*	*Drug and dose*	*When given (before or after defervescence)*	*Other therapy at time aspirin was given*
6	21	C	F	'Disprin' 10 gr.	After	Chloramphenicol, 3 g. per day
7	42	C	M	A.P. Cod., 2 tabs.	Before	None
8	39	C	M	A.P. Cod., 2 tabs.	Before	Penicillin and streptomycin
9	35	C	M	Aspirin, 30 gr. per day for 1 week	Before	None
10	22	C	F	Aspirin 10 gr.	After	Chloramphenicol

DISCUSSION

The transient symptoms which followed shortly after the administration of aspirin in the cases quoted leave little doubt that patients with typhoid fever may respond to small doses of aspirin with prostration, sweating, bradycardia and hypothermia. It is evident that not all patients with the disease react in this way to aspirin, nor is it necessarily the invariable reaction of any one patient given aspirin on more than one occasion (e.g. case 3).

It is possible that a similar reaction might occur in other febrile diseases, but I could find no record of this. The literature on the subject of antipyretics in typhoid fever, though scanty, indicates that a similar reaction may be produced by other antipyretic drugs.

The mechanisms by which the effects of aspirin in the disease are mediated are obscure and permit of only the most tentative hypotheses. It is conceivable that one part at least of the reaction may be attributable to an increased vagus and parasympathetic action, for bradycardia is a prominent feature of the acutely-ill phase and the general clinical state bears a certain

resemblance to that seen in the so-called 'vaso-vagal' attack.

Dr. Rachel Rabkin (1955) has suggested that the reaction is more likely to occur when the drug is administered to fasting patients, but there was nothing in this series to confirm or refute this suggestion.

A sudden fall in temperature with clinical deterioration during the course of typhoid fever is seen chiefly in association with the complication of haemorrhage or intestinal perforation, and awareness of the occurrence of this reaction following aspirin should obviate a mistaken diagnosis of one of these complications. As the administration of aspirin is not usually placed under the direct supervision of the medical staff, ward sisters should be warned of the dangers of giving aspirin to cases where typhoid fever is suspected.

The effects in all the cases in this series were transient and did not appear to influence the ultimate recovery.

The treatment of the patient during the acute phase of the drug-induced illness does not present a very great problem, since recovery may be expected after a matter of hours. One might expect sodium bicarbonate, through its action in hastening the elimination of aspirin, to be effective, but no convincing effect was seen when it was used in case 1.

SUMMARY

A series of 44 cases of typhoid fever is presented, 5 of whom illustrated a reaction that may follow the administration of aspirin to patients with this disease. This reaction, characterized by collapse, sweating, bradycardia and hypothermia, occurs shortly after the taking of the drug and subsides within a matter of hours.

Case histories are given.

My thanks are due to Dr. N. H. G. Cloete, Medical Superintendent of Groote Schuur Hospital, and to Professor J. F. Brock, Head of the Department of Medicine, for permission to publish these cases. In addition, I acknowledge, with gratitude, the generous advice and assistance of Dr. C. Merskey in the preparation of this paper.

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MOTOR NEUROPATHY APPEARING DURING THE COURSE OF TREATED TUBERCULOUS MENINGITIS

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and

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Isonicotinic acid hydrazide has now been in use for over 5 years in the treatment of tuberculosis in its various forms. Many toxic effects of the drug have been reported, including a number of cases of peripheral neuritis. This paper deals with a patient suffering from tuberculous meningitis who developed a lower-motor-neurone lesion during the course of treatment. The problems to decide were, firstly, whether the neuropathy was part of the disease or was due to one of the antituberculous drugs; secondly, whether or not to continue with the antituberculous therapy.

CASE REPORT

L.T., a 40-year-old Bantu male was admitted to Baragwanath Hospital on 10 December 1954, complaining of headache and cough for the previous week. On examination he looked ill and drowsy. He was afebrile and the rest of the physical examination was negative. Two days later his headache grew worse, his temperature rose to 103°F and he developed neck rigidity. Lumbar puncture revealed slightly turbid cerebrospinal fluid; the pressure was normal and there was no intrathecal block. Chemical and microscopic examination of the fluid showed 90 polymorphonuclears and 160 lymphocytes per c.mm., protein 13 mg. %, sugar 38 mg. %, chlorides 645 mg. %. He was regarded as a case of tuberculous meningitis and treated with 1 g. of streptomycin daily by intramuscular injection, and 150 mg. of Rimifon *tds* and 3 g. of para-aminosalicylic acid 4 hourly by the mouth. On 17 December tubercle bacilli were found in the cerebrospinal fluid on direct microscopic examination.

Over the next 4 weeks the patient gradually improved and his temperature settled. His cerebrospinal fluid still showed moderate pleocytosis, but as his general condition was good he was allowed out of bed.

About a month later, i.e. 2 months after admission and while still on therapy, he complained of low back pain and weakness in the legs. On examination he showed weakness of the flexors, extensors, abductors and adductors of the thighs, and the flexors and extensors of the knees and foot. Within 10 days wasting appeared and was marked below the knees. The knee and ankle jerks disappeared and he developed bilateral foot-drop. There was no sensory loss to light touch, pinprick or temperature; vibration and position sense were intact. Electrical stimulation of the nerves gave a reaction of degeneration. The cerebrospinal-fluid findings were 4 polymorphonuclears and 50 lymphocytes

per c.mm., protein 138 mg. %, sugar 48 mg. %, chlorides 705 mg. %.

The development of neuropathy in a patient who appeared so well clinically led to a suspicion that one of the drugs might be responsible for both the neurological signs and the persistent cerebrospinal-fluid pleocytosis. Accordingly all therapy was stopped for a week. Then all 3 drugs were administered in turn for 5 days. The cerebrospinal-fluid findings were unchanged after each trial period.

It was therefore concluded that there was no sensitivity to the drugs and that the pleocytosis indicated tuberculous activity. It seemed more likely that the neuropathy was a toxic effect of a drug than an expression of tuberculosis. Nevertheless it seemed to us more important to avoid recrudescence of the meningitis even if continued drug-therapy meant disabling the patient. We decided to give streptomycin and Rimifon with large doses of pyridoxine (150 mg. *tds*) and nicotinamide (100 mg. *tds*) as suggested by McConnel and Cheetham[1] and Biehl and Vilter.[2] On this regime, combined with physiotherapy, there was a gradual improvement in the patient's condition. At the end of 6 months he had little residual weakness, but his tendon jerks were still absent. The cerebrospinal-fluid findings at this time were 12 lymphocytes per c.mm., no polymorphonuclears, protein 86 mg. %, sugar 58 mg. %, chlorides 745 mg. %.

DISCUSSION

This patient while receiving 3 drugs developed a motor neuropathy and the question arose whether a drug was responsible. Except for one case of acquired hypersensitivity to PAS, streptomycin and penicillin, in which there were some sensory changes associated with pyrexia and skin rashes, the reported toxic effects of PAS do not include peripheral neuritis.

Although the neurotoxic effects of streptomycin are usually confined to the 8th cranial nerve, motor effects such as external ophthalmoplegia[3,4] and hemiplegia[5] have been reported following parenteral and/or intrathecal administration. Winters[5] considers that streptomycin causes organization of the subarachnoid exudate, dissolution of fibrin, and its replacement by actively proliferating fibroblasts and chronic inflammatory cells; tuberculous arteritis is transformed into a proliferative endarterial reaction causing severe stenosis and occlusion of vessels such as the spinal arteries, or those of the basal ganglia.

The toxic effects of INH are numerous, and the neurological reactions include psychosis, convulsions, euphoria, hyperreflexia, insomnia, disturbed vision, drowsiness and peripheral neuritis.[7,8,9,10]

The peripheral neuritis due to INH seems to follow a characteristic pattern.[8] Initially there is tingling of the fingers, followed by stiffness of joints; later the calves become tender, there is severe burning pain, and finally increasing weakness of the limbs. The reflexes are exaggerated at first and later disappear. Some authors[2,7,8] consider that the patient is more likely to develop neuritis if he has previously had INH and so is 'conditioned' to the drug. The onset of neuritis is variable and may take from 1 to 17 weeks[2,7,10].

Could the polyneuritis in this case have been due to thrombosis of the anterior spinal artery, arachnoiditis or pachymeningitis caused by the tuberculous meningitis? The first seems unlikely since it is usually associated with sensory loss and the signs of an upper-motor-neurone lesion. Arachnoiditis may cause multiple adhesions in the spinal canal which prevent free movement with respiration and thus interfere with the blood supply by pulling on vessels. Sometimes the arachnoid adhesions may enclose an encysted collection of cerebrospinal fluid which presents as a tumour. In either case there is usually some degree of blockage to the flow of cerebrospinal fluid, a situation which was not encountered in this case.

Tuberculous pachymeningitis only occurs as a result of extensive infection from tuberculous osteitis, which was not present in this case.

Treatment. The important practical problem in our case was whether to continue with antituberculous drugs or not. On the one hand the patient showed evidence of an active lesion, namely a cerebrospinal-fluid pleocytosis, which could not be left untreated. On the other hand he had developed a severe disability which, for all we knew, would not improve unless INH was withdrawn. We decided to persevere with INH, but added massive doses of nicotinic acid and pyridoxine, which are alleged to minimize the toxicity on nervous tissue.[1,2]

SUMMARY

A patient suffering from tuberculous meningitis, who was treated with INH, streptomycin and PAS, developed motor polyneuritis. Although it seems possible that INH was responsible for this lesion, the drug was not withdrawn and the patient made an excellent recovery.

We wish to thank Dr. J. D. Allan, Superintendent of Baragwanath Hospital for permission to publish this article.

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UNION DEPARTMENT OF HEALTH BULLETIN

Union Department of Health Bulletin. Report for the 7 days ended 3 May 1956.

Plague. Smallpox. Typus Fever. Nil.

At date of latest information there existed:

Plague: Nil.

Cholera in: Calcutta (India); Chittagong, Dacca (Pakistan).

Smallpox in: Rangoon (Burma); Phnom-Penh (Cambodia); Allahabad Bombay, Calcutta, Jodhpur, Kanpur, Madras, Pondicherry, Visakhapatnam (India); Chittagong, Dacca, Lahore (Pakistan); Saigon-Cholon (Viêt-Nam).

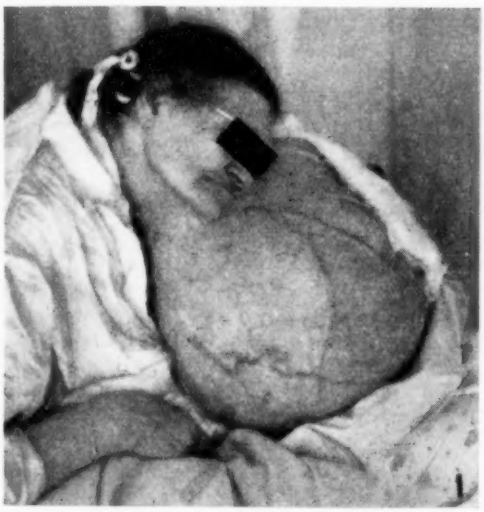
Typhus Fever in: Alexandria, Cairo (Egypt).

AN UNUSUAL TUMOUR

J. R. FRYLINCK, F.R.C.S.

Johannesburg

In July 1955 a European woman aged 63 was admitted to the Boksburg-Benoni Hospital because she had sustained a severe haemorrhage from the surface of a large tumour of her face. She stated that the tumour had been present for 30 years, showing a gradual increase in size over this period. She had objected to removal on religious grounds, and because a medical man had told her she would certainly die if this were attempted.



On examination the tumour seemed to be growing from the side of her face and neck, but no exact site of origin was discernible (Fig. 1). The tumour had pulled her head down and rested quite comfortably on her knees in the sitting position. Large veins coursed over its surface, and one of these had ruptured but had been easily controlled by pressure. The consistence was tensely cystic. The pre-operative diagnosis was that of a lipoma or possibly a liposarcoma. Some concern was felt from the physiological point of view about the removal of such a large growth. What portion of her circulating blood volume would suddenly be removed? Adequate transfusion arrangements were accordingly made.

The operation presented no difficulty. There were several large veins in the pedicle but haemorrhage was not troublesome. The centre of the tumour contained 3½ gallons of turbid fluid. The solid cortex weighed 25 lb, making the total weight of the mass 60 lb. The wound healed satisfactorily, but the patient has been left with facial paralysis on the side operated on, indicating that the tumour originated in the parotid gland.

The tumour tissue was submitted to Dr. B. Cohen, of the Oral and Dental Hospital, Johannesburg, who reported as follows:

'Sections were taken from solid portions of the tumour, from the surface lining the central cystic space and from an ulcerated area on the skin surface. The microscopic structure is that of a mixed salivary tumour. In the solid portion there are many areas simulating cystic changes which are due to distension of vessels, presumably a mechanical effect of the great weight of the growth. Necrotic change is widespread, and the tissue lining the central cyst-like chamber is composed entirely of necrotic debris. The surface erosion has the appearance of a benign ulceration. The over-all picture is of an uncomplicated mixed salivary tumour modified only by unusual heavy mechanical stresses.'

A second opinion was obtained from Dr. B. J. P. Becker, of the South African Institute for Medical Research, who concurred with Dr. Cohen's report.

PAMPHLETS FOR EXPECTANT MOTHERS

Dr. James Miller, obstetrician and gynaecologist of 4 Western Road, Port Elizabeth writes:

A feature of hospital obstetrics in certain areas of South Africa is the unnecessary material and foetal mortality and morbidity due to delay in reporting significant signs and symptoms. I have arranged pamphlets which I hope to have adopted locally, to cover these points, in simple English, Afrikaans and Xosa. These I am enclosing for reprinting in your *Journal* for the use of any one who wishes.

'I would like to acknowledge the help given by Dr. Ware and Dr. McLean the Superintendents of Livingstone and Provincial Hospitals, Port Elizabeth, and their secretaries Mrs. Brann and Mrs. Glover. Thanks for help in the translating go to Mr. Lanham of Rhodes, Dr. Steyn and Dr. Scholtz of Livingstone and Dr. Fick of the Provincial, Dr. Molefe, Mr. Barley and Mr. Jeffrey Booi; and thanks for criticism to the medical and nursing staff of the hospitals.'

SIX HINTS ON HOW TO HAVE YOUR BABY AND REMAIN HEALTHY

1. Report to a clinic or doctor when you feel you are going to have a baby, if:
   - (*a*) It is your first baby;
   - (*b*) you have had more than 5 babies;
   - (*c*) you are over 35 years old;
   - (*d*) a previous baby has died before, during or just after birth;
   - (*e*) your baby was born after you had been put to sleep by a doctor.

2. Go to the clinic or doctor when asked to attend, even if you feel well. Do as they say, they want to help you.

3. Go to the clinic or doctor even when not asked to attend, if:

(*a*) Your head aches and does not improve;
(*b*) you cannot eat and feel sick in the stomach;
(*c*) you bleed from your woman's parts, even if you have no pain;
(*d*) your water is not right, or there is leaking of water;
(*e*) there is swelling of your body.

4. When baby pains start, or blood comes from your woman's parts, or there is a sudden rush of water and anything hangs down, come to the hospital or call for your district nurse. You may have friends who want to have their baby at home. If it is their first baby, the sun should not set and rise on them twice, without help being called. If they have had babies normally before, the sun should not set and rise on them once. If a woman jerks her arms and legs when with child, her place is in hospital immediately. A woman should not feel like pushing or opening her bowels for more than 2 hours in a first labour, or 1 hour if she has had children—if the baby is not born she must go to hospital.

5. (*a*) The after-birth may take up to one hour to come out. Bleeding should not make you giddy or thirsty. There is to be slight bleeding for one month after the baby is born. This at first becomes brown, and then lighter again. (*b*) If twins are expected, the second one should start coming half an hour after the first.

6. Report to a doctor or a clinic after your baby is born:

(*a*) A sudden loss of red blood that flows freely or forms a large clot;
(*b*) a shivering attack;
(*c*) pain in the lower part of the body.

If you are asked to return to a clinic or a doctor in 6 weeks, or for your next baby, you must do this wherever you are living. This is especially important if there has been an operation.

PUBLIC HEALTH SERVICES AND THE TOURIST*

F. W. P. CLUVER, *M.P.C.*, M.B., CH.B., D.P.H.

Former Deputy Chief Health Officer of the Union

Why do people travel? What is it that attracts travellers to visit certain places? Why do people undertake holiday journeys? I think fundamentally, if not consciously, the travelling public is in search of health and recreation. Certainly the travelling public will not visit an area where they know the standard of health services is not what can be expected of a well-conducted local authority.

The proof of this was amply demonstrated last year in Durban, where the poliomyelitis threat was estimated to have resulted in a loss of some £200,000 in tourist trade. Another aspect is reflected in the annual report of the Durban Municipal Transport Board, which reports a loss of some £5,000 in its revenue as a direct result of the outbreak of poliomyelitis.

MALARIA

Many will remember the prevalence of malaria in Zululand before effective control measures abolished this scourge of our Low Veld areas. As one who was in charge of the campaign against malaria, I can speak with some knowledge of this subject. I remember in 1933, calling at a North Coast sugar mill, to find it standing idle, when it should have been crushing the cane waiting to be milled. The reason was that the personnel which should have been manning the mill was down with malaria, and it was simply not possible to operate the plant. In fact, in the early 'thirties' the very existence of the sugar industry was seriously threatened by the severe incidence of malaria in the sugar belt, and it was confidently asserted that you could have a sugar industry or malaria, but you could not have both.

The Union Government invited Professor Swellengrebel, a well-known malariologist, to come to South Africa and investigate the problem which, in the words of the late Dr. J. A. Mitchell, the first Secretary for Health and the creator of the Union Health Department, would, if solved, be equivalent to an extension of the boundaries of the Union. It would bring into production rich and well-watered areas, which at that time were lying untilled and neglected, and constituted an ever-present threat to the public health. The publication of the Swellengrebel report, which emphasized his discovery that only two species of anophelines (A. gambiae and A. funestus) were the causal agents of malaria in the Union, had far-reaching results.

The Province of Natal led the way and pioneered a system of control which later became world-famous. The technique used aimed mainly at the destruction of the adult mosquito vector by insecticidal spraying of human habitations. An enabling Ordinance was passed by the Natal Provincial Council in 1932, which established statutory bodies known as Malaria Committees in malaria areas. Later these Malaria Committee areas extended from the South Coast to the north-eastern boundary of the Province. These Malaria Committees were empowered to levy rates and under the guidance of the officers of the Union Health Department carried out a technique of control which was crowned with success. Similar control measures were pursued by the Union Health Department in the coastal Native Reserves.

Control consisted primarily in spraying Native huts and other human habitations with a pyrethrum-paraffin mixture (and, after the second World War, with DDT,) and the elimination as far as possible of vector breeding foci by drainage and saligna gum plantings. Incidentally saligna gum plantations have now become a major economic asset. The Amatikulu leper institution in Zululand, at which a seasonal outbreak of malaria occurred regularly amongst the patients, was effectively controlled by planting saligna gums in the surrounding valleys. These trees, which are very greedy drinkers, dried out the seepages in which the vectors bred. The work, done largely by the patients, effectively eliminated the vector-breeding water, and malaria is unknown there today. Incidentally, the gum plantations have resulted in a rich revenue (approximately £200,000) to the Union Treasury by supplying the raw material to the neighbouring pulp and paper factory.

It can be confidently asserted that the present prosperity of Zululand, with its rich sugar production, its profitable saligna industry, its cattle, citrus, and now its pineapple industry, would not have been possible had malaria not been brought under control. The enormous increases in land values are a spectacular witness to the effectiveness of the control of the scourge of malaria.

THE TOURIST INDUSTRY

But Zululand, in addition to its agricultural potentialities, also has a promising tourist industry, which is capable of considerable expansion. The Hluhluwe Game Reserve and the unique bird life in the St. Lucia estuary, as well as its fishing and other recreations, could attract many more tourists providing they are adequately catered for. In this, effective public health services play an important part.

I remember being urgently summoned to Bloemfontein in 1947 to meet the Royal train for consultation with the physician in attendance on the Royal Family. I was informed that the authorities in London were seriously perturbed because the Royal tour would traverse the malarial areas of Zululand during the malaria season; also because the Royal train would remain overnight at Gingindhlovu, which was reputed to be an intensely malarious area. I assured the Royal physician that there was no danger and that malaria control was so effective now that there was not the slightest risk to the Royal party. He seemed dubious, and

* A paper read at the 3rd Annual Conference of the Natal Regional Publicity Association, Eshowe, 20 April 1956.

informed me that he had just received a supply of Paludrin (at that time the latest malaria suppressive). I insisted that it was quite unnecessary to dose the Royal party with this suppressant drug. On their return to Durban from Zululand I had the honour of being presented to Their Majesties. The late King asked me if I was responsible for his having to swallow Paludrin tablets during his visit to Zululand. He also remarked that they had not seen a fly or a mosquito on their trip through Zululand and enquired what had happened to these insects. I believe that the fact that our Royal visitors were able to travel safely through Zululand during what used to be the malaria season served to put this part of the Union on the tourist map.

The Public Health Act of the Union, which, incidentally, is a monument to the late Dr. J. A. Mitchell, was passed by Parliament in 1919, after the devastating pandemic of influenza that followed the first World War. This act places the prime responsibility for the maintenance of public health on the local authorities. The larger local authorities, which include the chief cities of the Union, have large, experienced and well-qualified staffs, under the direction of experienced medical officers of health. The larger local authorities are well-staffed and well-equipped, and able to maintain a high standard of health in the communities which they serve. They are subsidized by the Union Health Department in respect of their qualified medical officers of health, health inspectors, district nurses, and other approved personnel, and function virtually as autonomous bodies within their areas.

The smaller local authorities, such as Local Boards, Health Committees and Village Management Boards, which have small rate incomes cannot, of course, maintain a health staff comparable with that of the larger centres. Very often they have only a part-time medical officer of health and one full-time health inspector. These bodies can, and do, call on the staff of the Union Health Department for guidance and expert information. It is often surprising to see what excellent services are rendered in the promotion of health in these small centres by the available staff.

OTHER PREVENTABLE DISEASES

Dr. Cluver then went on to speak of other preventable diseases that are the concern of health authorities. He mentioned the enteric fevers, dysenteries and diarrhoeas; tuberculosis; food poisoning and tapeworm; plague, typhus, yellow fever and trypanosomiasis; and bilharzia disease. In this connection he discussed the importance of water supplies, sewerage, milk and food supplies, insect pests, and bilharzia control. He referred also to the duties of health authorities in relation to nutritional diseases and mental disorders.

CONCLUSION

The values of human health are not to be measured in monetary terms alone. The preamble of the World Health Organization constitution states that the enjoyment of the highest attainable standard of health is one of the fundamental rights of every human being. There is a moral, not merely a financial, issue involved. It is clear, however, that it will be easier to obtain the support needed for an effective health programme if it can be shown that such a programme will not only enrich the individual human life but will also bring tangible economic benefits to the community which invests in health. *Prevention is not only better than cure, it is also cheaper than cure.*

No centre which wishes to encourage visitors or to maintain an existing tourist industry dare neglect essential health services. There is no single factor which can so easily destroy a tourist industry as an outbreak of infectious disease; and this could often have been prevented if the authorities had adopted measures now available to enlighten communities.

RESEARCH FORUM, UNIVERSITY OF CAPE TOWN

PRIMARY ALDOSTERONISM*

L. EALES, M.D., M.R.C.P.
and
G. C. LINDER, M.D., F.R.C.P.

The first case of primary aldosteronism reported in South Africa was presented and is to be reported in detail elsewhere. The case was in the person of a Cape Coloured female aged 32 years, who presented with a 5-year history of recurring temporary paralyses of the limbs and severe tetany with paraesthesia. On examination there was evidence of chronic renal disease, viz. persistent hypertension, slight but constant proteinuria, polyuria, and a fixed urinary specific gravity. The urine was always alkaline or feebly acid.

Biochemical investigation of the serum showed a hypokalaemic alkalosis with intermittent hypernatraemia. The hypokalaemia was temporarily reversible on heavy potassium loading. The electrocardiogram showed changes consistent with hypokalaemia.

Extensive studies during the past 3 years showed that there was a gross depletion in total body-potassium and that this depletion was related to an excessive urinary wastage of potassium. An enlarged (R) adrenal was demonstrable by perirenal insufflation of air by the pre-sacral route (Dr. D. Brink) and an increased urinary aldosterone excretion was found (Dr. B. Lewis). Reversal to normal of the many abnormalities followed the operative removal on 30 November 1955 (Professor J. H. Louw) of a bright-orange tumour weighing 28 grams. The histological appearance of the tumour was that of a benign cortical adenoma.

Among the more interesting aspects discussed was the observation that the kidneys did not show the 'vacuolar nephropathy' of potassium depletion in marked degree, possibly on account of the prolonged potassium repletion she had undergone. Although the finding of organisms in the urine in similar cases has led to a diagnosis of chronic pyelonephritis, it is probable that potassium deficiency in producing severe tubular lesions acts as a locus minoris resistentiae for infection, but the abnormal urinary pH may be an additional contributory factor.

Follow-up to date shows that she is well. Renal function, as determined by clearance techniques, had deteriorated before operation and has since improved, although the glomerular filtration rate has not yet returned completely to normal. Despite this, the abnormally high urinary potassium wastage has been abolished; on clinical testing there is no proteinuria; and a maximal urinary specific gravity of 1,025 has been obtained after 12 hours of fluid deprivation.

* Summary of a paper read at a Research Forum meeting held at Groote Schuur Hospital, Cape Town, on 10 April 1956.

NEW PREPARATIONS AND APPLIANCES : NUWE PREPARATE EN TOESTELLE

'*Tes-Tape*' (*Urine Sugar Test Tape, Lilly*). Eli Lilly and Co. announces this test tape (originated by Dr. Albert S. Keston, New York) for the rapid colorimetric percentage determination of the sugar in urine by the patient. It is supplied in a handy plastic dispenser that resembles those used for cellophane adhesive tapes. Each dispenser holds enough tape for approximately 100 tests. The tape is made of a fine-grain filter paper, and is impregnated with enzymes.

The patient dips one end of a 1½-inch strip of tape into a specimen. If glucose is present in the urine, two enzymes—glucose oxidase and peroxidase—act to produce changes in the colour of the yellow tape. These changes range from light green to deep green or blue depending on the amount of glucose present. The test is specific since glucose oxidase exerts its catalytic action only on glucose. A colour chart on the dispenser gives a direct comparison reading. The chart is coded: 0 (0%, + (1/10%), ++ (1/4%), +++ (1/2%), and ++++ (2% or more). These concentrations in general coincide with the 0, 1+, 2+, 3+ and 4+ the diabetic patients may be used to recording. The convenience of 'Tes-Tape' allows even a busy person to run tests during the working day. For travel the advantages are obvious. Hospital technicians who must run tests on a number of patients will find 'Tes-Tape' a time-saver.

* * *

Ergo-Rondase. Evans Medical Supplies, in announcing the introduction of Ergo-Rondase, a dual pack containing one ampoule of ergometrine maleate, 0·5 mg. and a vial containing 333 i.u. of Rondase (Hyaluronidase, Evans), state than an intramuscular injection of Ergo-Rondase acts as quickly as an intravenous injection of ergometrine alone. This enables the nurse or midwife to exploit to the full the beneficial effects of ergometrine.

PASSING EVENTS : IN DIE VERBYGAAN

Dr. Joseph Wolpe of Johannesburg has been invited to be a Fellow at the Center for Advanced Study in the Behavioral Sciences at Stanford, California, USA, during the year 1956-57. He intends to leave South Africa with his family about the middle of July. The Center, which is sponsored by the Ford Foundation, each year brings together 50 scientists who are selected on the merits of their published contributions in various fields of human behaviour. Dr. Wolpe plans, while at Stanford, to continue his researches on the mechanisms that lead to neurosis and on the development of improved methods of psychotherapy.

* * *

Dr. S. Berman has been elected to the Fellowship of the Royal College of Physicians of London. Dr. Berman is full-time Head of the Department of Neuropsychiatry at the Groote Schuur Hospital and lecturer in Neuropsychiatry to the University of Cape Town Medical School.

* * *

Dr. Lennox Eales and *Dr. Clarence Merskey* have been appointed by the Rockfeller Foundation to travelling Fellowships in Medicine. They are both full-time physicians in the Department of Medicine, Groote Schuur Hospital, and lecturers in the Department of Medicine, University of Cape Town. Dr. Merskey's field of research is in haematology and Dr. Eales' research in the field of kidney diseases. Dr. Eales will leave early in September for 6 months, and Dr. Merskey about the middle of December also for 6 months. Both are proceding to America.

* * *

The Henry Simpson Newland Prize in Surgery. This prize is awarded by the Federal Council of the British Medical Association in Australia every 3 years to the writer of the essay adjudged to be the best on a surgical subject. The next award will be made in 1958, the subject of the essay being 'Factors Influencing the Prognosis in Acute Intestinal Obstruction'. The prize is a money award of £100, together with a medal.

The dissertation is to be based on personal observation and experience, and the essay must be typewritten or printed in English. It must be distinguished by a motto, and accompanied by a sealed envelope containing the name and address of the author, and having on its outside the corresponding motto. It must not exceed 50,000 words.

The competition is open to any graduate of any medical school within the British Commonwealth. Essays must be delivered to the General Secretary, Federal Council of the British Medical Association in Australia, 135 Macquarie Street, Sydney, New South Wales, not later than 23 November 1957. The prize essay must be submitted forthwith to the Editor of the *Medical Journal of Australia.*

* * *

Summer School in Health Education. The Central Council for Health Education, London, is organizing a Summer School at Stoke Rochford, near Grantham, Lincolnshire, England, from 14 to 24 August. The course is for doctors, nurses, and other social workers engaged in community health services. It will provide an intensive practical training in modern methods of health teaching.

Participants will be housed in single study-bedrooms at Stoke Rochford, which is a large country mansion, and a number of social activities and visits to places of interest will be arranged. The theme of this Summer School will be Teamwork and Techniques in Health Education. The inclusive fee for the course, including residence, will be £21 0s. 0d., of which £2 2s. 0d. is payable on registration. Applications should be made to the Medical Director, Dr. J. Burton, B.A., M.R.C.P., D.P.H., The Central Council for Health Education, Tavistock House, Tavistock Square, London, W.C. 1.

REVIEWS OF BOOKS : BOEKRESENSIES

MEDICAL OFFICE PROCEDURES

Office Procedures. By Paul Williamson, M.D. Pp. 412+ix with illustrations. £5 6s. 3d. Philadelphia & London: W. B. Saunders Company. 1955.

Contents: 1. Ear, Nose and Throat. 2. Eye. 3. Musculo-skeletal System. 4. Gynecology. 5. Obstetrics. 6. Urology. 7. Proctology. 8. Pediatrics. 9. Minor Surgery. 10. Internal Medicine. 11. Psychological Testing. 12. Anesthesia. 13. Physiotherapy. 14. The Small Laboratory. 15. Roentgenography.

In this reviewer's opinion the title of this book of more than 400 pages is not quite accurate—it belies its contents. The nature of the procedures described, more often than not, cannot be undertaken in an ordinary surgery. As a rule one simply has not the time and the proper facilities needed. They are more likely to be found in a small, staffed and well-equipped clinic. The author seems to rely chiefly on the use of local anaesthesia for the accomplishment of most of his 'minor' procedures, which might rather be classed in the 'major' category! Appendicectomy is a case in point, and there are many others in the book.

True, we doctors are assumed to know how to carry out minor office procedures. These are many and varied in nature, depending most often on the individual practitioner's knowledge and aptitude, and in acquiring them the 'hard way' has often been the best teacher. Few of us can benefit fully from other people's experience without ourselves 'trying it out' first. In general practice, one invents useful aids and short cuts, especially when one has to dispense with assistants and expensive instruments. It is at such times, that one experiences the truth of Wordsworth's words, 'Man needs but little here below....'

This book would be a veritable *vade mecum* to doctors in outlying clinics. It gives much sound advice and many a timely warning. In the section on the Musculo-skeletal System the author makes the point, all too easily forgotten in our days, that 'every back that hurts is not a ruptured disc'. He might have added, '*nor is it a displaced vertebra*'—a very easy, very tall, and more often than

not erroneous, diagnosis, and one which is expensive to the patient. Moreover, throughout the book the author warns his readers that certain conditions should not be undertaken outside a hospital.

The whole section on Minor Surgery is excellent, as well as the one on the Musculo-skeletal System. Those on Proctology and Urology are somewhat too concise but very helpful.

The author discusses sinus trouble at some length, but does not seem to refer to the more common *and more troublesome* condition of frontal neuritis; similarly subdeltoid bursitis is discussed, but rheumatic affection of the shoulder musculature has been overlooked, a condition which is much more common in everyday practice. The treatment described for 'baseball finger', and the illustration accompanying it, seem to be incorrect; hyperextension and not flexion seems indicated. On page 64 directions for blocking the brachial plexus tell one to direct the needle point toward the fourth lumbar instead of, presumably, thoracic vertebra. In the section on Obstetrics, reference is made only to the Aschheim—Zondek and the Friedman tests for pregnancy but not to the frog test. These are, however, minor points.

The work as a whole is excellently presented; the sketches are simple and clear and appear in great profusion throughout the text. They are so placed as to form an integral part of the text itself. This combination of text and illustrations is so far the best that one has come across. The printers have certainly done a good job, too.

This work should prove to be a real help to anyone starting a practice, as well as to house surgeons.

G.C.A.v.d.W.

MODERN OBSTETRICS AND GYNAECOLOGY

Modern Trends in Obstetrics and Gynaecology (*Second Series*). Edited by Kenneth Bowes, M.D., M.S. (Lond.), M.B., Ch.B. (L'pool), F.R.C.S., F.R.C.O.G. Pp. 407+xv with 170 illustrations. 67s. 9d. London: Butterworth & Co. (Publishers), Ltd. South African Office—Butterworth & Co. (Africa) Limited, Durban. 1955.

Contents: 1. The Development and Shape of the Female Pelvis. 2. The Mechanism of Myometrial Function and its Disorders. 3. The Cellular Components of the Human Ovary. 4. The Ovarian Vasculature and Ovarian Functions. 5. Ovulation About the Time of the Menopause. 6. The Influence of Oestrogens on the Formation of Uterine Tumours. 7. The Sex Chromatin and its Application to Errors in Sex Development. 8. Pain in Gynaecological Conditions. 9. The Metabolic Changes Associated with Operation. 10. The Pathology of Anuria in Obstetrics. 11. A Hypothesis of the Aetiology of Toxaemia of Late Pregnancy. 12. The Circulation in Pregnancy and Toxaemia of Pregnancy. 13. Aspects of the Reproductive Physiology of the Post-Partum Period. 14. Maternal Obesity. 15. The Forceps Operation in Modern Obstetrics. 16. Acute Abdominal Conditions in Pregnancy and Puerperium. 17. The Establishment and Maintenance of Respiration in the Newborn. 18. The Early Diagnosis of Intersex. 19. Emergency Surgery of the Newborn. 20. Radiology in Obstetrics and Gynaecology. 21. The Post-Menopausal Endometrium. 22. Rare Conditions Associated with Amenorrhoea. 23. Antibiotics in the Treatment of Genital Tuberculosis in the Female. 24. Premenstrual Tension. 25. The Treatment of Carcinoma of the Cervix Uteri. 26. Vulval Atrophy and Leukoplakia. Index.

The second series of 'Modern Trends' is not another edition of the first book—the contents are wholly different—but should be regarded as being supplementary to the first series.

For the first time South Africa has contributors in this series, viz. Professor O. S. Heyns (The development and shape of the female pelvis) and Professor Ian Donald, now of Glasgow, (The establishment and maintenance of respiration in the newborn). These articles illustrate the wisdom of policy of getting experts to present their views on parts of their specialities in which they are particularly interested.

In reviewing this book one is immediately struck by the widening scope of Obstetrics and Gynaecology. In particular the range of Endocrinology is particularly brought out; no fewer than 18 out of the 26 chapters have an endocrinological basis.

The little-known subject of the ovarian vasculature and its influence in the production of pathological changes in the ovary, is well documented by Reynolds. The contentious issue of oestrogens and uterine neoplasia is well handled by the late Harold Burrows. Barr writes on his method for determining the chromosomal sex of an individual. The importance of this determination in every case presenting with primary amenorrhoea should be realized. Theobald writes a stimulating chapter on pain in gynaecological conditions, and Sheehan's article on anuria should help in elucidating this problem. Mastboom's hypothesis on the aetiology of the toxaemias of late pregnancy is a masterpiece. Bishop writes on rare conditions associated with amenorrhoea. It is a pity that the term 'gonadal dysgenesis' is not substituted for the less satisfactory one 'ovarian agenesis'. After all, most of these cases are really males. It is gratifying to see that the ovarian hyperthecosis syndrome has at last found its way into an English textbook on Gynaecology. It is certainly not so rare as the author supposes. Ryden, after extensive experience with the problem of genital tuberculosis, gives his views on the modern treatment of the condition. Wallace makes a plea for the abolition of the word 'kraurosis' and suggests the commendable term 'primary atrophy of the vulva'.

This second series is an excellent book and it should be as well received as its predecessor.

W.H.M.

CORRESPONDENCE : BRIEWERUBRIEK

'THE ANTERIOR CHEST WALL SYNDROME'

To the Editor: Myron Prinzmetal and Massumi[1] recently described what they consider to be a clinical entity, 'The Anterior Chest Wall Syndrome' which appears to be a bad case of putting the cart before the horse. Briefly, it consists of a tender and painful anterior chest wall occurring commonly in those who have previously suffered a coronary infarction, and occsionally in those who have not.

We are all familiar with the tender condition of the intercostal muscles, which may be painful on movement, which commonly follows an infarction, and I believe that most of us consider it to be the somatic manifestation of an anxiety state. Painful muscle-tensions commonly accompany a 'tension state'.

However, while Prinzmetal recognizes the common coexistence of an underlying psychological imbalance he does not acknowledge this to be the basic cause. Thus he remarks that 'cancerophobia is a not uncommon outcome of this condition'. Surely, particularly where there has been no coronary trouble, the cancerophobia is the cause. At the same time he admits that 'emotional stress seems to have an aggravating effect'.

Courses of ACTH and X-ray therapy (given without placebo controls) were successful in curing this condition. While it is possible that as a stress syndrome the physiology of the affected muscles might benefit from such therapy, the primary attack must be with psychotherapy and, even if the physical methods of treatment are adopted, such therapy must be explained as being directed against the secondary effects of anxiety and stress.

The cases referred to indicate that the condition is occasionally iatrogenic in origin, which must be the result of placing soma before psyche. Should such an approach be made to this condition in future, the result will be that many people will receive expensive courses of therapy as a primary attack, whereas such treatment should only be used as a last resort.

Paul Wood,[2] in describing the effort syndrome, views this pain (for it is the same one) in the proper perspective.

J. Kelman Drummond

1. Prinzmetal, M. and Massumi, R. A. (1955): J. Amer. Med. Assoc., **159**, 3.
2. Wood, P. (1950): *Diseases of the Heart and Circulation*, 1st ed., p. 537. London: Eyre and Spottiswoode.

DISPENSING BY DOCTORS

To the Editor: I trust that the medical profession will firmly resist the present attempt that is being made by chemists to prevent doctors from doing their own dispensing.

By dispensing, doctors learn to know the medicines they use and prescribe for their patients. This intimate knowledge of drugs is of great value to doctors and helps them to dispense medicines which are effective and whose taste and side-effects are not unacceptable to the patient.

I wonder how chemists would react if the medical profession were to ask them to give up counter-prescribing for the numerous patients who consult them every day for their various complaints and those of their children and babies. It is a well-known fact that many patients first seek the aid of a chemist before consulting a doctor, and it is only when the medicines prescribed by the chemist fail to relieve or cure the patient that he calls in the doctor. Furthermore, chemists treat many patients with chronic diseases which are at present incurable.

If chemists think it is wrong for doctors to compete with them in dispensing, do they think it is right for chemists to compete with opticians, jewellers, photographers, and general dealers by testing eyesight, and selling spectacles, jewellery, watches, cameras, films, field-glasses, fountain pens, baby foods and other side-lines?

Finally, the basic reason why doctors should continue to dispense their own prescriptions is that the vast majority of patients in South Africa are poor non-Europeans who, at present, are able to obtain a medical consultation and examination and receive medicine for fees varying in different parts of the country from 3s. 6d. to 10s. 6d. If doctors were to cease dispensing, these poor patients would have to pay a fee to the chemist as well as the doctor. This would raise the cost of medical treatment, which would be to the detriment of the health and welfare of these patients and their families, because there would remain less money for food, clothing and shelter.

General Practitioner

2 May 1956

‚VAN DIE REDAKSIE'

Aan die Redakteur: In die rubriek ‚van die Redaksie' in die *S.A. Tydskrif vir Geneeskunde* van 28 April 1956, lees ons in die eerste paragraaf: ‚Die *siektebeheer* van akute *verstopping van die hart* is grotendeels toegespits op die voorkoming en behandeling van die komplikasies. Omtrent die helfte van die pasiënte wat die ramp self oorleef sal *rustig* herstel. By die ander word die herstel bemoeilik deur talle komplikasies—hartversaking, *aritmie*, *propvorming*, hart*breuk* en skok'. En verder: ‚Terloops kan dit weer beklemtoon word dat matige dosisse digitalis wel by *hartverstopping* gebruik kan word. Die *aritmies* word volgens hulle oorsprong behandel; die meeste *boesem-aritmies* word doeltreffend met digitalis behandel, terwyl die wat in die *kamers* ontstaan met *chinidien* of *procaine* amied beheer word'.

Alhoewel ek geen taalkundige is nie, en ook goed besef dat u baie moeilikhede met Afrikaans as tegniese taal het, wil ek in alle beskeidenheid voorstel dat die volgende vertaling (want dis duidelik dat u uit die Engels vertaal) meer leesbaar en boweal verstaanbaar sal wees.

‚Die *beheer* van akute *myokardiale infarksie* is grotendeels toegespits op die voorkoming en behandeling van komplikasies. Omtrent die helfte van die pasiënte wat die ramp oorleef sal *sonder verdere insident* herstel. By die ander word herstel bemoeilik deur talle komplikasies—hartversaking, *aritmia*, *tromboembolisme*, hart*ruptuur* en skok'.

En verder: ‚Terloops kan dit weer beklemtoon word dat matige dosisse digitalis wel by *myokardiale infarksie* gebruik kan word. Die *aritimias* word volgens hulle oorsprong behandel, die meeste *supravetriekulêre aritmias* word doeltreffend met digitalis behandel, terwyl die wat in die *ventriekels* ontstaan met *kinidien* of *prokain* amied beheer word'.

J. Knobel

Posbus 70
Dewetsdorp
2 Mei 1956

LOW BACK PAIN: A SUGGESTION

To the Editor: I would like to suggest a possible cause for some cases of low back pain on the left side. As an occasional sufferer myself, it has occurred to me that this might be due to the repeated working of the clutch with the left foot, which is necessary because of the frequent change of gears which the motorist has to execute in an urban area. It is conceivable that this, over a lengthy period, might be responsible for strain of the muscles and ligaments in the lumbo-sacral area.

I. Hendler

18, Mullins Road
Malvern East
Johannesburg
6 May 1956

TREATMENT OF ORAL THRUSH, HERPETIC STOMATITIS, MALIGNANT MALNUTRITION AND GASTRO-ENTERITIS IN INFANTS

To the Editor: As a result of experience in the Out-patient Department in the Department of Child Health at Groote Schuur Hospital, Cape Town, I beg to make the following observations:

(1) A 1% aqueous solution of gentian violet is a well-known and effective treatment for oral thrush; it is useful in most forms of stomatitis, but it is often ineffectively applied. By means of cotton wool on the end of sticks, brushes, feathers and the like one may at best apply the drug to the anterior parts of the oral cavity; at worst one may traumatize an already injured mucous membrane. By contrast 3-5 minims of the solution *dropped directly* into a baby's mouth will spread effectively and without trauma, applying the drug to the entire oral cavity and pharynx.

(2) Broad-spectrum antibiotics and penicillin, which inhibit the growth of bacterial flora, thus encouraging the growth of fungus and of virus, are contra-indicated in oral thrush and in herpetic stomatitis. Antibiotics should be used with caution in these conditions, and are probably needed only in the presence of secondary bacterial infection indicated by enlargement or tenderness of cervical lymph-glands. In this event sulphonamides are the chemotherapeutic agents least likely to have deleterious effect.

(3) Vitamins B and C seem to have value in the treatment of oral thrush and of herpetic stomatitis, but are completely wasted —as are all non-protein-containing foods—in the treatment of malignant malnutrition (kwashiokor, nutritional oedema) unless correction of the *basic protein deficiency* in this condition is undertaken, e.g. by the forced feeding of milk. Angular stomatitis and the skin lesions of kwashiokor are healed completely by adequate protein feeding alone without any additional vitamins whatever; without adequate protein feeding, however, other measures are vain.

(4) Streptomycin and penicillin by injection are useless therapeutic agents for infantile gastro-enteritis.

Cases which have been incorrectly treated for not inconsiderable periods of time are referred daily to hospital. A wider appreciation of these few simple points would, I think, save many infants much suffering.

Basil Goldschmidt
Registrar

Department of Pediatrics
Groote Schuur Hospital
Cape Town
5 May 1956

RADIOLOGISTS AND FAECAL IMPACTION

To the Editor: It is to be brought to the notice of radiologists that they are the cause of many anal fissures and painful inflammatory attacks of haemorrhoids. The cause is neglect on their part to give full and implicit instructions to the patient on how to get rid of the radio-opaque barium introduced into the alimentary tract.

Some of these patients are already sufferers from chronic constipation and the clearance of the barium must be a careful and planned procedure of massive doses of liquid paraffin with milk of magnesia and, in the resistant cases, the supplementing with oil and water enemas. By this treatment the harmful sequelae can be completely prevented.

Every one of these patients must be treated as a case of threatened impaction in order to avoid the unnecessary pain and suffering which at present is occuring far too often.

Stephen Eisenhammer

34 Moray House
Cor. Jeppe and Smal Streets
Johannesburg
4 May 1956